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PRINCIPAL INDICATIONS FOR SPLENECTOMY DURING CHILDHOOD

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THE THERAPEUTIC value of splenectomy in certain blood dyscrasias has become more widely recognized during the past twenty-five years. Despite the fact that splenic function is not fully understood, sufficient progress has been made as a result of both clinical and hematologic investigation to permit of definite conclusions as to treatment. Splenic dysfunction occurs in congenital hemolytic icterus, thrombocytopenic purpura and Banti's syndrome. In such diseases splenectomy is especially beneficial.

TABLE 1.—*The Indications for Splenectomy in Children*

Disease	Number of Cases
Thrombocytopenic purpura	22
Congenital hemolytic icterus	20
Banti's syndrome	7
Splenic panhematocystopenia	1
Hypoplastic anemia	1
Sickle cell anemia	1
Traumatic rupture of the spleen	1
Pseudohemophilia	1
Gaucher's disease	1
Total	55

It is the purpose of this paper to present the principal indications for splenectomy in children and to evaluate results obtained. Splenectomy was performed on 55 children at the University Hospital between September 1933 and August 1946. The youngest child was 30 days old and the oldest 12 years. Among the indications for operation were congenital hemolytic icterus (20 children), thrombocytopenic purpura (22 children) and the Banti syndrome (7 children). In the remaining 6,

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splenectomy was performed for rarer disturbances, including splenic panhematocytopenia, traumatic rupture, sickle cell anemia, hypoplastic anemia, Gaucher's disease and pseudohemophilia. The diagnoses in the 55 cases are presented in table 1.

Congenital hemolytic icterus is a chronic blood dyscrasia characterized by microcytic anemia, increased fragility of the erythrocytes, reticulocytosis, acholuric jaundice and splenomegaly as well as the occasional occurrence of "crises." Although the essential diagnostic features are now well recognized, the fundamental cause remains obscure.

The severity of the symptoms varies greatly, even differing in the same patient from time to time. Frequently the onset of acute symptoms, tending toward the critical phases, is preceded by an infection of the upper respiratory tract or some other minor ailment. The chief complaints may then be weakness, fatigue and dyspnea due to anemia or merely a vague abdominal discomfort due to the splenomegaly. The hemolytic crisis may be ushered in by an attack of nausea and vomiting, with fever.

TABLE 2.—*Presenting Symptoms of Congenital Hemolytic Icterus*

Symptoms	No. of Cases	Per Cent
Jaundice	14	70
Weakness	8	40
Pallor	7	35
Splenomegaly	6	30
Nausea and vomiting	2	10
Chills and fever	2	10
Abdominal pain	1	5

The presenting symptoms of 20 children with congenital hemolytic icterus are listed in table 2.

The severity of the hemoclastic crisis may be such that the patient may become critically ill within a few hours. With extensive hemolysis the erythrocyte count may drop as low as 600,000 and the hemoglobin content to 2 Gm. per hundred cubic centimeters. The clinical picture resembles that of sudden severe loss of blood with extreme weakness, dyspnea and even semicoma. This critical state was present in 6 of the 20 children (30 per cent). Emergency splenectomy was carried out as the therapeutic procedure of choice. It proved to be a lifesaving measure in 5. The child who did not recover died four hours postoperatively. The severe critical condition had become irreversible, and the emergency splenectomy proved of no avail. The rationale for emergency splenectomy is based on the hypothesis that the spleen is the major hemolytic agent.[1]

1. Curtis, G. M.; Doan, C. A. and Wiseman, B. K.: Splenectomy for Hemoclastic Crises, Ann. Surg. **104**:892, 1936.

Doan, Curtis and Wiseman [2] have shown that a definite and immediate postoperative rise of the erythrocyte count and the hemoglobin content ensues after splenectomy during the acute crisis.

The age of the infant with congenital hemolytic icterus should be no contraindication to splenectomy. Our youngest patient was 30 days old.

The untreated infant in the critical phase of congenital hemolytic icterus may not survive. The time of choice for splenectomy is as soon as the diagnosis is made. In the treatment of patients who show but little external manifestations it is common practice to defer splenectomy with

TABLE 3.—*Age, Sex, Mortality and End Results Among Twenty Children Subjected to Splenectomy for Congenital Hemolytic Icterus*

Patient	Sex	Age	Duration of Symptoms	Date of Operation	Preoperative Red Blood Cell Count	Postoperative Red Blood Cell Count (Latest)	Follow-Up
G. P.	F	4 yr.	1 mo.	9/16/33	1,020,000	4,200,000	Improved
A. M.	M	8 yr.	4 yr.	11/16/33	1,980,000	3,500,000	Improved
N. B.	F	7 yr.	Since birth	8/20/35	3,303,000	4,220,000	Improved
N. D.	F	7 yr.	1.5 yr.	3/24/39	3,020,000	4,520,000	Improved
W. H.	M	12 yr.	Since birth	6/ 8/39	3,500,000	7,120,000	Improved
F. W.	F	3 yr.	7 mo.	11/ 3/40	790,000	.....	Died 4 hours postoperatively
T. N.	M	11 yr.	2 wk.	4/17/41	750,000	4,800,000	Improved
J. C.	F	9 mo.	3 mo.	6/ 2/41	1,420,000	4,020,000	Improved
W. R.	M	3 yr.	Since birth	9/ 4/41	2,870,000	4,250,000	Improved
J. G.	M	2 yr.	Since birth	12/ 1/41	3,360,000	3,590,000	Improved
H. G.	M	7 yr.	Since birth	12/ 1/41	3,240,000	4,420,000	Improved
I. S.	F	7.5 wk.	Since birth	1/22/42	790,000	4,330,000	Died of internal obstruction 4 months postoperatively
I. C.	F	10 yr.	1 wk.	2/18/43	1,030,000	4,230,000	Improved
D. G.	F	9 yr.	1 wk.	2/24/44	2,990,000	4,610,000	Improved
G. P.	M	12 yr.	2 yr.	6/28/44	3,810,000	4,810,000	Improved
L. P.	M	8 yr.	1 yr.	6/29/44	4,180,000	5,100,000	Improved
D. S.	M	30 days	Since birth	9/ 1/44	1,610,000	4,030,000	Improved
M. G.	F	8 yr.	2 wk.	11/12/45	1,800,000	3,240,000	Improved
S. S.	F	4 yr.	2 wk.	1/16/46	4,000,000	3,780,000	Improved
S. P.	F	7 yr.	1 wk.	7/17/46	3,130,000	4,690,000	Improved

the hope that it may not be necessary. If this course is pursued, the disease may change from one of extreme chronicity to one which is acute, and the result may be fatal. Under careful preoperative and postoperative management surgical intervention can be carried out with success in a high percentage of cases. Splenectomy for congenital hemolytic icterus effects a "cure" by reestablishment of the hematopoietic equilibrium. This in turn will remove the burden on other organs, such as the heart and biliary system, and evidences of wider disturbed function will disappear.

Table 3 presents the findings and the end results in the 20 children subjected to splenectomy for congenital hemolytic icterus.

After splenectomy in 20 children with congenital hemolytic icterus there was 1 death, a mortality of 5 per cent. The death followed an

2. Doan, C. A.; Curtis, G. M., and Wiseman, B. K.: The Hemolytopoietic Equilibrium and Emergency Splenectomy, J. A. M. A. **105**:1567 (Nov. 16) 1935.

emergency procedure during an acute critical phase. With the surgical risk so low and the beneficial results so immediate and permanent in nature, splenectomy should be advised in every case of proved congenital hemolytic icterus.

Although knowledge concerning the nature of thrombocytopenic purpura has developed gradually since it was first described by Werlhof [3] in 1775, there is as yet no complete agreement as to the etiologic factors involved or as to the mechanism by which these factors act. Since 1916, when Kaznelson [4] first suggested splenectomy as a mode of treatment, sufficient experience has been gained to justify fully the removal of the spleen. Of greatest importance is the careful and thorough examination essential for the establishment of the correct diagnosis. In our series the hematologic studies were conducted in the Department of Research Medicine by Dr. Charles A. Doan, Dr. Bruce K. Wiseman and Dr. Claude-Starr Wright, who gave me permission to use their data.

The symptoms usually presented were evidences of hemorrhage, ecchymotic or petechial, as well as bleeding from mucous membranes such

TABLE 4.—*Presenting Symptoms in Twenty-Two Children with Thrombocytopenic Purpura*

Symptoms	No. of Cases	Per Cent
Subcutaneous hemorrhages (ecchymoses or petechiae)	20	91
Bleeding from mucous membranes	16	73
History of bruising easily	7	32
Weakness	2	9

as the nose, mouth, rectum or vagina. If the bleeding were prolonged, evidence of secondary anemia was noted. One third of the patients gave a history of bruising easily.

The presenting symptoms of the 22 children with thrombocytopenic purpura are listed in table 4.

With the diagnosis once established, the question of the optimum time for splenectomy and of whether it should be performed in acute as well as chronic forms of the disease arises. Many have proposed that splenectomy be done only in cases in which the disease is classed as "chronic." Doan, Curtis and Wiseman [2] have shown that splenectomy is not contraindicated in properly selected cases of thrombocytopenic purpura, even in the presence of an acute crisis, provided adequate preoperative blood transfusions are given. Delay in removing the spleen may result in a fatality because of uncontrollable hemorrhage, either internal or

3. Cohn, I., and Leman, I. I.: Splenectomy As a Treatment for Purpura Hemorrhagica (Thrombocytopenic Purpura, Kaznelson) with Report of Case and Review of the Literature, Surg., Gynec. & Obst. **38**:596, 1924.

4. Kaznelson, P.: Wien. klin. Wchnschr. **29**:1451, 1916; cited by Andrus, W. DeW., and Holman, E. W.: Splenectomy in Various Blood Disorders, Ann. Surg. **109**:64, 1939.

external. Two of the patients under consideration were treated with preliminary supportive measures under careful observation. Despite medical management the condition of the children became progressively worse. Emergency splenectomy in both instances proved efficacious in restoring the children to a state of well-being. The remaining 20 were operated on during the interval of quiescence. There was 1 fatality after splenectomy, a mortality of 4.5 per cent. Death was due to intraperitoneal hemorrhage from a lacerated vein in the splenic pedicle. The 21 children followed in the clinic for from seven months to five years have been symptom free and have greatly improved. Table 5 presents the findings and the end results.

TABLE 5.—*Findings and End Results in Twenty-Two Patients Subjected to Splenectomy for Thrombocytopenic Purpura*

Patient	Sex	Age	Duration of Symptoms	Date of Splenectomy	Platelets, Preoperatively	Platelets Postoperatively (Latest)	Follow-Up
D. H.	F	4 yr.	1½ wk.	3/23/38	None seen	.....	Died
T. V.	F	12 yr.	4 mo.	5/13/41	16,440	832,000	Improved
D. N.	F	12 yr.	4 days	9/24/41	None seen	1,323,420	Improved
R. C.	M	8 yr.	6 mo.	10/ 7/41	One seen	286,520	Improved
W. W.	M	8 yr.	1 yr.	2/13/42	11,120	221,760	Improved
C. S.	F	5 yr.	2½ mo.	8/22/42	3,860	2,120,800	Improved
N. B.	F	12 yr.	6 mo.	9/ 1/42	4,940	329,080	Improved
J. S.	F	5 yr.	2½ mo.	11/17/43	None seen	865,200	Improved
J. N.	M	4 yr.	2 yr.	7/ 1/44	9,840	603,360	Improved
D. G.	M	5 yr.	3 yr.	7/ 8/44	10,720	664,240	Improved
W. B.	M	2 yr.	3 wk.	10/ 4/44	7,300	641,540	Improved
C. M.	F	4 yr.	4 yr.	1/ 6/45	15,640	1,494,900	Improved
P. W.	M	4 yr.	2 yr.	6/ 2/45	16,840	925,160	Improved
M. W.	F	8 yr.	4 mo.	7/16/45	9,280	317,190	Improved
J. J.	F	5 yr.	3 wk.	7/25/45	17,760	586,520	Improved
R. K.	M	10 yr.	2 yr.	7/25/45	16,320	275,940	Improved
M. M.	F	10 yr.	1½ mo.	7/28/45	9,760	272,880	Improved
A. M.	F	6 yr.	3 yr.	8/17/45	8,840	760,640	Improved
D. D.	M	18 mo.	2 mo.	8/27/45	8,040	900,000	Improved
M. S.	M	12 mo.	2 wk.	3/28/46	39,600	583,680	Improved
E. B.	F	12 yr.	3 yr.	4/26/46	15,640	772,800	Improved
V. R.	F	4 yr.	1 yr.	7/ 3/46	12,840	1,515,000	Improved

On consideration of the brilliant immediate results following splenectomy together with the restoration to normal living of the great majority of children with thrombocytopenic purpura it may be said that this operation has contributed the greatest advance to the therapy of the disease.

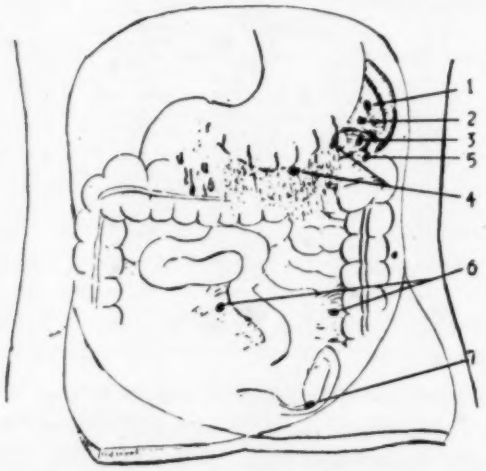
In 7 of the patients the condition was diagnosed as Banti's syndrome. The changes observed in the spleen after splenectomy were compatible with those commonly found. The surface was usually covered with vascular adhesions and the capsule greatly thickened. The splenic vein was tortuous in its course and greatly enlarged.

All 7 had splenomegaly, anemia and leukopenia. They complained mainly of weakness and repeated gastric hemorrhages and, less often, of epistaxis, purpura and pain in the left upper quadrant of the abdomen. Table 6 presents the findings and the end results.

Of the 7 patients with Banti's syndrome subjected to splenectomy, 1 died in the hospital, an operative mortality of 14.3 per cent. The operative risk may be increased by severe perisplenic adhesions rich in an increased collateral circulation as well as by the fragility and size of

TABLE 6.—*Findings and End Results in Seven Children Subjected to Splenectomy for Banti's Disease*

Patient	Sex	Age, Yr.	Duration of Symptoms	Date of Splenectomy	Preoperative: Red Blood Cell Count	Preoperative: White Blood Cell Count	Postoperative: Red Blood Cell Count	Postoperative: White Blood Cell Count	Follow-Up
M. W.	F	10	3 yr.	4/26/34	2,420,000	2,100	.....	.....	Died on the sixth postoperative day
D. S.	M	12	2 yr.	1/20/41	1,210,000	950	3,630,000	6,500	Improved
D. G.	M	7	1 yr.	10/ 3/42	3,220,000	3,300	4,410,000	11,200	Recurrence of hematemesis
R. P.	M	7	4 yr.	10/28/43	3,860,000	1,650	1,270,000	900	Unimproved
D. H.	F	7	4 yr.	4/23/45	3,600,000	1,350	4,030,000	2,350	Improved
D. K.	F	2	3 days	1/16/46	1,990,000	3,650	3,570,000	16,100	Improved
P. S.	M	8	1 yr.	3/16/42	2,260,000	3,450	2,430,000	6,900	Unimproved



Areas where accessory spleens may be found—*1*, the hilar region; *2*, the splenic pedicle; *3*, the retroperitoneal region surrounding the tail of the pancreas; *4*, the great omentum, especially along its attachment to the greater curvature of the stomach; *5*, the splenocolic ligamentary attachments; *6*, the mesentery of the large and small intestines; *7*, in the female, the left adnexa.

the dilated and tortuous veins in the splenic pedicle. The spleen is commonly adherent, and a collateral circulation of its upper pole may develop, which adds to the operative difficulties. Splenectomy for the Banti syndrome meets with the greatest success if confined to the early stages of the disease. The operation is also justified in the later stages since the patient may also experience severe discomfort because of the size of the spleen.

Curtis and Movitz [5] have stressed the surgical significance of the accessory spleen in congenital hemolytic icterus and thrombocytopenic purpura. Failure to remove the accessory spleen at the time of splenectomy may result in a recurrence. Accessory spleens were found in 2 of the 22 children with thrombocytopenic purpura and in 13 of the 20 children with congenital hemolytic icterus.

DISCUSSION

DR. ROBERT S. DINSMORE, Cleveland: I feel sure that Dr. Van Buren would have had many more points to present had he had more time, but he has made some very important points which should be emphasized.

First of all, in regard to children with thrombocytopenic purpura, a surgeon is often called in consultation as to the advisability of performing a splenectomy. The surgeon should realize that he is only seeing one third of the cases which the hematologist has under his care. My colleagues and I have analyzed 120 cases and have operated in 28 of these. We have advised splenectomy in some others, but the percentage is low. We feel that the percentage should be higher, probably around 40 per cent. As a consultant you are completely at the mercy of the medical man, but as a consultant you should be accurate in your diagnosis. You must know the clinical history and be absolutely sure that you do not have a child victim of some other platelet-depressing disease. You must know all the chronic illnesses and all the infections that the child has had and all the drugs that he has taken, so that you will know you are not dealing with some other type of disease. None of these patients should be operated on without study of the bone marrow, which, of course, takes time.

Another point which I wish to bring out is that unless the response of the platelets is marked the prognosis is bad. We have a group in which the platelet count has gone up but has subsequently decreased. We have another group in whom the platelet count did not go up, and they are all right.

When the diagnosis of hemolytic icterus is made, the patient should be operated on. If there are any other children in the family who have any of the symptoms of this hemorrhagic disease, you are justified in requesting that those children have an operation. I have operated on a father and four of seven children for this condition.

DR. GEORGE M. CURTIS, Columbus, Ohio: Patients with congenital hemolytic icterus should not have transfusions prior to splenectomy. Such a procedure only adds fuel to the fire, and renal complications may result. On the other hand, the patient with thrombocytopenic purpura should have transfusions preoperatively. There is no similar mechanism in this disease. If you have a patient in the critical phase of congenital hemolytic icterus, splenectomy should be done at once and without preoperative transfusion. The spleen is usually moderately enlarged, and with the use of epinephrine hydrochloride and massage it will return many corpuscles to the circulation. Moreover, you can have isotonic solution of sodium chloride and blood ready to flow into a vein so that just as soon as the splenic pedicle is clamped the transfusion may be begun because then the hemolytic mechanism is out. The converse is true in thrombocytopenic purpura —you can continue to give transfusions to the patient before, during and after the splenectomy.

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5. Curtis, G. M., and Movitz, D.: The Surgical Significance of the Accessory Spleen, Ann. Surg. **123**:276, 1946.

PLASTIC SURGICAL REPAIR OF FACIAL PARALYSIS

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IT IS NOT the purpose of this presentation to discuss new methods for the correction of facial paralysis but to familiarize this group with some of the methods and results obtained in the treatment of injury of facial nerves.

SELECTION OF CASES

It is difficult to estimate with consistent accuracy in which cases the facial paralysis may tend to regenerate spontaneously and, when this does occur, what degree of regeneration may be anticipated. Naturally, lesions secondary to removal of extensive mixed tumors of the parotid gland or acoustic neuromas may be looked on as permanent. Those following trauma such as skull fractures, injury from mastoid operations or bullet wounds may arise from edema around the nerve rather than from actual division of the nerve trunk. If the edema is slight, spontaneous response may be noted within a short time, but with more extensive injuries evidence of some return of function of the facial muscles may be noted well over one year after the original injury. In these instances of apparent late regeneration it has also been experienced that the return of function is only partial and never complete. In fact, in a few cases in which operation has been performed at a late date there probably has been a little function present for a long time, which has been masked by the overaction of the normal side before it has been controlled by a supporting operation on the paralyzed side.

Consequently, in view of the experience of my colleagues and me with cases of facial paralysis in which it cannot be ascertained whether or not the nerve has actually been severed, we believe that some type of surgical repair should be undertaken if considerable spontaneous return of function has not become manifest within three to four months after injury. This suggestion is made because only partial spontaneous return of nerve function can be anticipated beyond this point, and

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supplementation of function in a partially paralyzed face by one of the supportive operations will eliminate much of the overaction of the normal side and hence permit the injured side to function to the greatest possible degree.

SURGICAL REPAIR

The operative methods for the correction of facial paralysis may be classified under the following groups: (1) neurosurgical repair, (2) mechanical support with fascia lata, (3) transplants of temporal and masseter muscle and (4) combination of mechanical support with transplants of fascia and muscle.

With the possible exception of those with injury of facial nerves following radical resection of contiguous neoplasms, all patients with lesions of the facial nerve in whom satisfactory return of function has not been shown within three months should be given the benefit of neurosurgical consultation before any method of plastic surgical repair is undertaken. The nerve trunk divided by a lacerated wound should be united by prompt primary suture of the two ends.[1] Wounds in which there is loss of nerve substances, and these are usually within the facial canal, can frequently be repaired with a nerve graft to fill in the hiatus.[2]

Another type of neurosurgical procedure is that of nerve substitution.[3] Anastomosis of the distal end of the divided facial nerve to the hypoglossal or spinal accessory nerve has been executed many times, but the net results are not too acceptable because the associated movements involved in trying to move the facial muscles are awkward and frequently more conspicuous than the original facial paralysis. In consequence, operations for anastomosis of nerves are losing popularity, and procedures giving rise to better function are more favored.

Support of the paralyzed face with autogenous strips of fascia lata was first advocated by Kirschner[4] and Lexer,[5] but refinements of their original methods have followed during the ensuing years. In general, the early procedures involved the subcutaneous transplantation of strips of fascia from an incision in the temporal area down to the angle of the mouth, the corner of the nose and beneath the lower eyelid. When fixed to these points they were sutured at their upper ends to the temporal

1. Bunnell, S.: Suture of the Facial Nerve Within the Temporal Bone, Surg., Gynec. & Obst. **46**:7 (July) 1927.

2. Bunnell, S.: Surgical Repair of the Facial Nerve, Arch. Otolaryng. **25**: 235 (March) 1937.

3. Ballance, C. A., and Duel, A. B.: The Operative Treatment of Facial Palsy, Arch. Otolaryng. **15**:1 (Jan.) 1932. Duel, A. B.: Clinical Experiences in Surgical Treatment of Facial Palsy by Autoplastic Nerve Grafts, ibid. **16**: 767 (Dec.) 1932.

4. Kirschner, M.: Der gegenwärtige Stand und die nächsten Aussichten der autoplastischen freien Fascien—Übertrang, Beitr. z. klin. Chir. **86**:5, 1913.

5. Lexer, E.: Wiederherstellungschirurgie, Leipzig, J. A. Barth, 1920.

fascia, enough tension being used to hold the paralyzed face at a normal level. This method did not produce any activation, but a fair cosmetic result was obtained, and the patients invariably stated that they felt more comfortable when the sagging was eliminated.

It would be of interest at this point to mention the use of tantalum wire or mesh as a substitute for the use of fascia strips. The former is of course simpler from the standpoint of the procedure involved. However, tantalum wires have frequently cut through from pressure necrosis, and there always remains the potential danger of reaction[6] to any type of foreign material over a period of time. Since living tissue (i. e., strips of autogenous fascia) has proved satisfactory over many years there seems to be little excuse for the use of material of the foreign body type such as tantalum.

At the present time, when a corrective operation is selected that involves the use of strips of autogenous fascia lata, certain refinements are added to the original Kirschner technic, the most important of which follow: The first modification concerns the point of insertion of the lower strip of fascia at its distal point. A better support of the lower lip is obtained when the terminal end of the fascia is carried completely across the lower lip beneath the vermilion border to the opposite or normal angle of the mouth, where it is sutured to the orbicularis oris muscle. When secured at this point, the temporal end of the fascia is drawn taut and the strip sutured to the opposite or paralyzed angle of the mouth at a point which holds it at a level. This method of insertion of the lower strip gives better support to the lower lip and, being attached to the normal angle of the mouth, aids in preventing the active side from overpulling.

The second modification of the early technic involves the point of attachment of the upper or proximal ends of the transplanted strips of fascia lata. Instead of being secured to the temporal fascia, they are now fixed to the underlying temporal muscle. Since this muscle is supplied by the fifth cranial nerve and is still active, a certain amount of motion can be transmitted down the strips of fascia and a slight contraction of the side of the face obtained when the patient bites. The amount of activity obtained however is variable and has some limitations because of the length of fascia involved. If the proximal ends become fixed to adjacent tissues, the amount of motion that may be produced distally is naturally diminished.

In order to improve the results obtained by simple fascial support and to gain further activation, the procedure had been supplemented by various types of operations utilizing muscle pedicles, chiefly through

6. McIndoe, A. H.: Personal communication to the author.

the contributions of Gillies,[7] Sheehan,[8] Bruner,[9] Adams [10] and Owens.[11] The use of muscle pedicles alone does not ordinarily support and correct the sagging of the face. Hence, if the asymmetry is overcome by the use of support with fascia lata some activation can then be added by transplantation of one or more muscle pedicles.

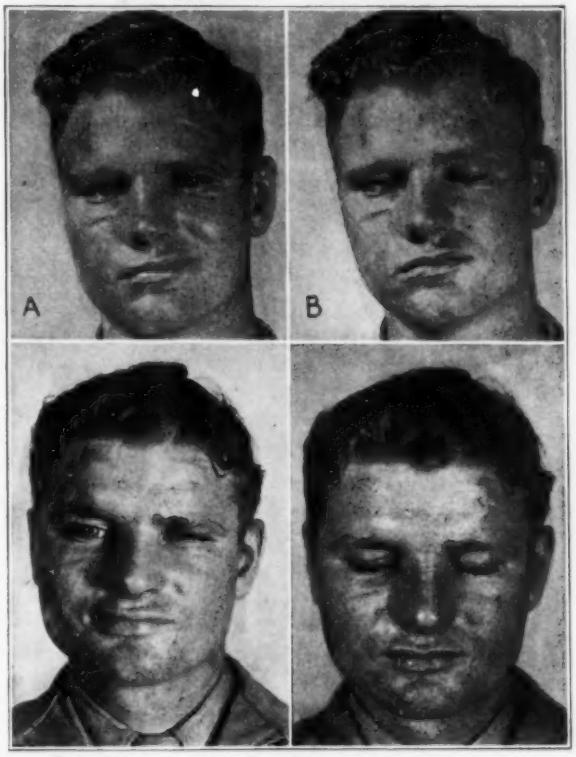

Fig. 1.—*A*, facial paralysis with pronounced asymmetry, *B*, inability to close right eyelids; *C*, paralyzed side has been supported with strips of autogenous fascia lata only; *D*, right lower eyelid, with paralytic ectropion, elevated with strip of fascia lata only so that palpebral fissure can be closed.

7. Gillies, H. D.: Experiences with Fascia Lata Grafts in the Operative Treatment of Facial Paralysis, Proc. Roy. Soc. Med. **27**:98 (Aug.) 1934.

8. Sheehan, J. E.: The Muscle: Nerve Graft in Unilateral Facial Paralysis, S. Clin. North America **15**:471 (April) 1935.

9. Bruner, H.: Ueber plastiche Operationer Facialislähmung, . Arch. f. klin. Chir. **140**:85-100, 1926.

10. Adams, W. M.: The Use of the Masseter, Temporalis and Frontalis Muscles in the Correction of Facial Paralysis, Plast. & Reconstruct. Surg. **1**:216 (Sept.) 1946.

11. Owens, N.: The Use of the Masseter Muscle in Effecting Facial Expression Following Complete Paralysis of the Seventh Nerve, Tr. Am. Soc. Plast. & Reconstruct. Surg. **2**:25 (Jan.) 1947.

For the angle of the mouth, a bundle of the temporal muscle may be turned downward or one from the anterior border of the masseter muscle mobilized upward and forward. For the eyelids, two bundles, one for the upper and one for the lower lids, are turned forward from the anterior surface of the temporal muscle. In any of these transfers of muscle the muscle bundle proper is never long enough to reach the point to which it is to be secured. Consequently, additional length is obtained by suturing a strip of either fascia lata or temporal fascia to the distal end of the muscle and finally into the terminal points at the angle of the mouth and to the periosteum of the base of the nasal arch or inner canthal ligament, where the lid strips are anchored. With these muscle bundles secured to the angle of the mouth and around the eyelids, the patient can obtain some activation by learning to bite down, i. e., to contract the temporal and masseter muscles, when he wishes to smile.

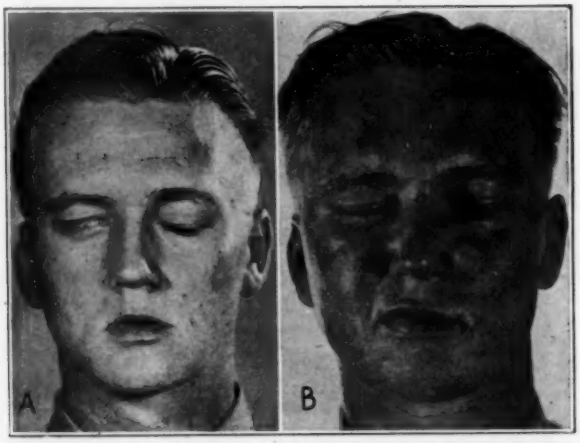

Fig. 2.—*A*, injury to right facial nerve following skull fracture. Note inability to close right eyelids. *B*, facial paralysis corrected by support with fascia lata and transplants of temporal muscle to right angle of mouth and beneath right lower eyelid. Closure of eyelid and contraction of right side of face produced by biting on right side, i. e., flexion of transplanted strips of temporal muscle.

An additional procedure has been utilized by Adams [10] which permits some elevation of the paralyzed brow. This is accomplished by transferring a pedicle from the medial border of the normal frontalis muscle to a point where the lower border of the pedicle is secured subcutaneously to the medial portion of the opposite or paralyzed side of the forehead.

It should be pointed out that in the performance of any operation utilizing muscle pedicle a reasonably large mass of muscle should be used. Failure to observe this may lead to disappointing results, since a narrow pedicle may undergo atrophy from lack of sufficient blood supply to maintain its viability.

In persons who have had facial paralysis of long standing one frequently observes considerable stretching of the overlying facial skin. In situations of this type a better end result can be obtained if the redundant skin is excised such as is done in a "face-lifting" operation, the procedure, of course, being confined strictly to the paralyzed side.

A further point of practical importance deals with the level at which the transplanted fascia or muscle pedicles are placed. Fascia should not be inserted too superficially or a permanent skin furrow will be noted along its course beneath the skin. Likewise, when a large pedicle of temporal muscle is turned down to the angle of the mouth, a large bulge in the cheek will be made where the mass crosses the zygomatic arch. This can be obviated by resecting a segment of the zygomatic arch through which the muscle pedicle may drop and thus eliminating the swelling.

SUMMARY

1. All patients with facial paralysis should be given the benefit of neurosurgical consultation before a plastic operative procedure is decided on. Nerve suture and nerve grafts give the most ideal results in selected cases. The operations of nerve anastomosis are not desirable because the associated movements involved in trying to move the face are usually more conspicuous than the original facial paralysis.

2. The simplest and most satisfactory plastic surgical correction is obtained by supporting the paralyzed face with strips of autogenous fascia lata. The use of tantalum in any form is not recommended for this purpose.

3. Some activation of the fascia-supported face can be obtained by supplementing the operation with one of the muscle-transplanting procedures. In the transferral of a muscle pedicle, it is urged that a bundle of sufficient size to maintain its viability be employed in order to obviate atrophy and hence failure.

4. The success or failure of the paralyzed face to gain motion after a muscle-transplanting operation depends largely on the intelligence of the patient and his willingness to practice movements of facial expression before a mirror until he has educated himself on how to utilize the muscles in their new locations.

TONUS OF THE BLADDER DURING SPINAL "SHOCK"

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IN THE past, many workers have reported that the urinary bladder is in an atonic state immediately after injury to the spinal cord. Holmes,[1] Walker[2] and Fearnsides[3] observed men with injuries of the spinal cord during the first World War and came to the conclusion that the detrusor muscle was always in a lax condition after injury to the medulla spinalis. More recently it has been stated by Munro and Hahn[4] and by Riches[5] that there is complete atonicity or flaccidity of the detrusor muscle during the period of spinal "shock" (a condition following transection, contusion, concussion or edema of the spinal cord).

Valk[6] studied 112 cases of acute injury of the spinal cord during the second World War, and in no case did he observe flaccidity of the detrusor muscle in patients in whom drainage was carried out immediately after the injury. Experimentation on animals by Kolb, Langworthy and Lewis[7] has indicated that section of the cord as well as of the sacral roots did not produce an immediate stretching of the detrusor muscle.

Utilizing the tetraethyl ammonium ion, we[8] have demonstrated that chemical blockade of the sympathetic and parasympathetic ganglions in human beings did not affect the tonicity of the bladder or its ability to maintain an approximately constant intravesical pressure with varying volumes of fluid. The cystometrograms in figure 1 show the effect of

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From the Department of Surgery, University of Michigan Medical School.

Read at the fourth annual meeting of the Central Surgical Association, Chicago, Feb. 22, 1947.

1. Holmes, G.: Brain **56**:383, 1933.

2. Walker, J. T.: Lancet **1**:173, 1917.

3. Fearnsides, E. G.: Brain **40**:150, 1917.

4. Munro, D., and Hahn, J.: New England J. Med. **212**:229, 1935.

5. Riches, E. W.: Brit. J. Surg. **31**:135, 1943.

6. Valk, W. W.: Personal communication to the authors.

7. Langworthy, O. R.; Kolb, L. C., and Lewis, L. G.: Physiology of Micturition, Baltimore, Williams & Wilkins Company, 1940.

8. Nesbit, R. M.; Lapides, J.; Valk, W. W.; Sutler, M.; Berry, R. L.; Lyons, R. H.; Campbell, K. N., and Moe, G. H.: J. Urol. **57**:242, 1947.

intravenous administration of tetraethyl ammonium bromide on an uninhibited neurogenic bladder. It will be noted that tonicity and the ability to accommodate to increasing volumes of fluid at the same intravesical pressure are unchanged although the uninhibited contractions of the detrusor muscle have been abolished and apparent vesical capacity increased.

Careful analysis of the papers concerned with this problem during the first World War leads one to the conclusion that the patients investigated were usually seen days or even weeks after injury to their spinal cord. Catheterization was usually not accomplished during the period immediately after injury; the patients had retention of urine with resultant overdistention of the bladder for days before urinary drainage was established, if it was established at all.

The present investigations were made on the tonicity of the detrusor muscle in cases of spinal "shock." Cystometrograms were made of bladders that were catheterized and maintained on inlying catheter

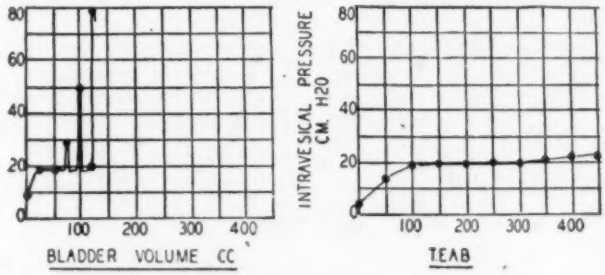

Fig. 1.—Effect of intravenous administration of tetraethyl ammonium bromide on an uninhibited neurogenic bladder.

drainage immediately after injury to the spinal cord. Spinal anesthesia produces the same effects as those exhibited in spinal "shock," namely, loss of spinal reflexes, with a resultant flaccidity of the skeletal musculature below the site of the lesion or that portion of the cord affected by the anesthetic. On the basis of this fact, it was decided to carry out observations on the tonicity of the bladder under spinal anesthesia.

METHODS

Cystometry was utilized in evaluating the status of the tone of the bladder. The apparatus consisted of a two-eyed soft rubber catheter, which was connected to a water manometer calibrated in centimeters. Distilled water from a graduated reservoir was allowed to run into the bladder at an approximately constant rate of 30 cc. per minute, and the intravesical pressure was recorded.

Observations were made on 6 patients with injuries of the spinal cord and on patients under spinal anesthesia. Experimentation on animals was also performed in which cystometrograms were obtained on dogs under light spinal anesthesia (in which pentobarbital sodium was used) immediately after section and crushing of the sacral cord and two hours after section of the cord.

RESULTS

Figure 2 *A* is a cystometrogram obtained on a dog weighing 13 Kg. under anesthesia produced by pentobarbital sodium. It can be observed that the bladder accommodates normally to increasing volumes of fluid at a water pressure of approximately 12 cm. The capacity of the bladder is 75 cc., at which volume the voiding contraction occurred. A laminectomy was then performed and procaine hydrochloride injected through the dural sheath into the area surrounding the spinal cord. In figure 2 *B* can be seen the reaction of the bladder to increasing volumes of fluid under spinal anesthesia. It will be noted that the curve is exactly the same as that in figure 2 *A* with the exception that the voiding contraction is absent and instead there is a straight line relationship between volume of fluid and intravesical pressure (*a* to *b*). This occurs when the bladder is stretched beyond its capacity and merely illustrates the reaction of the elastic connective tissue of the wall of the bladder to increasing volumes of fluid. Spinal anesthesia has eliminated the segmental reflex at sacral levels 2, 3 and 4, and thus no motor impulses

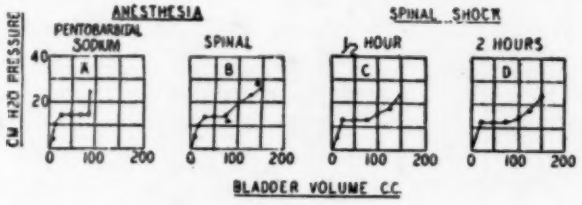

Fig. 2.—Cystometrogram made on a female dog weighing 13 Kg.

can be transmitted to the detrusor muscle to cause it to contract when the capacity of the bladder is reached. At point *b* there was leakage around the urethral catheter.

The dog's spinal cord was then transected at the level of the second lumbar spinal segment, and the portion below the transection was thoroughly crushed. After this procedure a cystometrogram was obtained; this is illustrated in figure 2 *C*. Figure 2 *D* demonstrates the reaction of the bladder two hours after spinal transection. It will be observed that the cystometrograms are exactly the same as those obtained while the animal was under the influence of spinal anesthesia. The tone of the detrusor muscle as well as its ability to maintain an approximately constant intravesical pressure with varying volumes of fluid was not abolished or decreased.

Cystometrograms made on a human subject before and during spinal anesthesia are demonstrated in figure 3. It will be seen that the curve prior to spinal anesthesia exhibits uninhibited contractions and indicates a voiding contraction at a vesical volume of 250 cc. Spinal anesthesia

abolished both the uninhibited contractions and the voiding contraction, although again it did not affect the tone of the detrusor muscle or its power of accommodation. Apparently the vesical capacity was 300 cc., since the straight line relationship could be observed when the volume was increased from 300 to 400 cc. of fluid.

Figure 4 demonstrates the cystometrograms of the bladder of a 46 year old patient who fell 40 feet (12 M.) from the top of an embank-

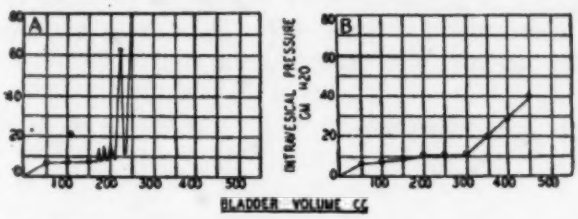

Fig. 3.—Uninhibited neurogenic bladder. *A*, before spinal anesthesia; *B*, during spinal anesthesia.

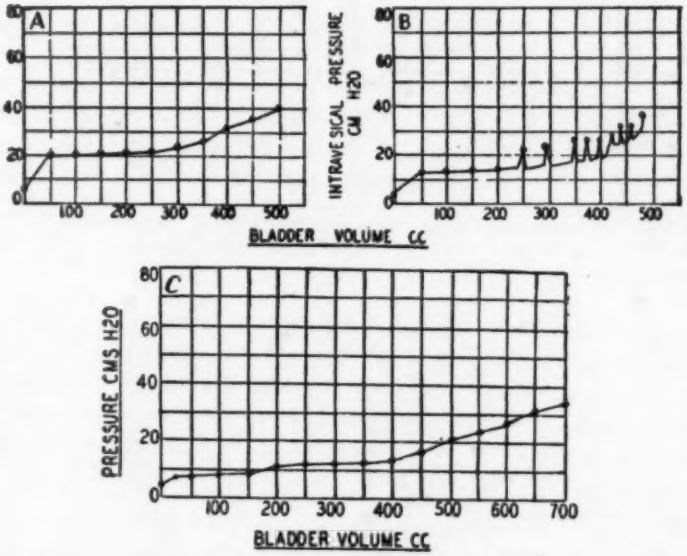

Fig. 4.—Cystometrogram made on a patient with subluxation of the spine at the sixth cervical vertebra, with transverse myelitis. *A*, four days after onset of spinal shock; *B*, partial recovery seven weeks after injury; *C*, effect of sepsis on spinal reflex activity thirteen weeks after injury.

ment and sustained a subluxation of the spine at the junction of the sixth and seventh cervical vertebrae, with an apparent compression fracture of the body of the seventh. Physical examination disclosed a complete flaccid paralysis from the level of the shoulders caudad.

Respiration was entirely diaphragmatic in type. Shortly after the diagnosis was made, ice tong traction was applied and continuous drainage with an inlying urethral catheter begun. Cystometric examination (fig. 4 *A*) was performed four days after the initial injury. It will be noted that tone and accommodation of the bladder appear normal. The voiding contraction is absent. Seven weeks after injury the bladder, coincident with the appearance of skeletal reflexes, began to demonstrate a response to segmental reflex activity, as indicated by the small uninhibited contractions in figure 4 *B*. These contractions, as well as skeletal reflex activity, disappeared at the end of the thirteenth week after injury (fig. 4 *C*). It is believed that the disappearance of activity in the severed spinal cord was due to the generalized sepsis and pyrexia produced in the patient by large infected bedsores. This opinion is in accord with the observations of Head and Riddoch,[9] who stated that "attacks of septicemia, whatever the origin, greatly delay the establishment of automatic micturition and all febrile conditions tend to diminish the responsive power of the spinal cord."

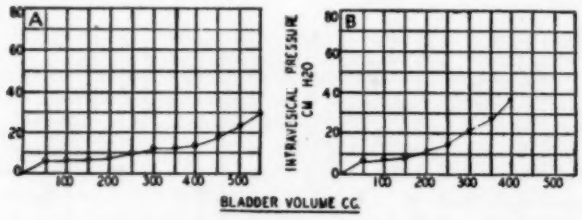

Fig. 5.—Cystometrogram made on a patient with a compression fracture of the first lumbar vertebra. *A*, seven hours after onset of spinal shock; *B*, five weeks after injury.

The cystometrogram in figure 5 *A* was obtained seven hours after the onset of spinal shock in a 60 year old carpenter who fell from the roof of a building and received a compression fracture of the first lumbar vertebra. Again it can be seen from the cystometric examination that tone and accommodation of the bladder are within normal limits. In figure 5 *B* it can be observed that reflex activity had not returned five weeks after injury.

COMMENT

It is well to interpret the term "tonus" before further discussion is attempted. Cobb and Wolff [10] in a review article on the tonus of skeletal muscle made a plea that the term "tone" be discarded so far as the description of the state of tension in striated muscle is concerned. It was suggested that more specific terms such as "standing reflex," "pos-

9. Head, H., and Riddoch, G.: Brain **40**:188, 1917.

10. Cobb, S., and Wolff, H. G.: Muscle Tonus, Arch. Neurol. & Psychiat. **28**:661 (Dec.) 1932.

tural reflex" and "righting reflex" be substituted for the term "tone." The development of tension in striated muscle is independent of voluntary innervation but dependent on stimuli of some type coming to the muscle from the central nervous system; it is a reflex phenomenon. If the nerve going to one of the skeletal muscles is cut, the muscle becomes elongated and flaccid. This flaccidity and loss of tension in striated muscle are observed in the first stage of acute injury of the spinal cord, which is characterized by the loss of spinal reflexes and vaguely termed spinal "shock."

It has been commonly observed that smooth muscle will maintain much of its tension or resistance to extension under conditions of complete denervation. Some smooth muscle will even exhibit rhythmic contractions under the same conditions. These characteristics are utilized in differentiating between skeletal and plain muscle.[11] It is thus difficult to visualize the state of flaccidity or atonicity occurring in bladders of patients suffering from acute injury of the spinal cord since one of the properties of smooth muscle is that it maintains its tonus despite loss of the spinal reflexes or its extrinsic nerves. Some workers [6] believe that the higher centers play a role in determining the tonus of the detrusor muscle. We [8] have demonstrated that chemical blockade of the autonomic ganglions did not decrease the tone of the bladder or its ability to accommodate to increasing volumes of fluid at the same intravesical pressure although the voiding contractions were abolished. Further observations in the present investigation have demonstrated that spinal anesthesia and spinal "shock" produced no essential change in tonicity or accommodation of the detrusor muscle although there was a complete absence of any voiding contraction. From these observations it would appear that normally there are no tonic efferent discharges being mediated by the parasympathetic or motor nerves to the detrusor muscle. It is believed that the lower motor neurons (at the level of the second, third and fourth sacral spinal segments) send their discharges down the pelvic nerve only for the purpose of initiating and maintaining the emptying contraction of the bladder. Normally the lower motor neurons are inhibited from discharging impulses by the corticospinal tracts, which are under voluntary control. When the bladder is filled to that capacity which creates the desire to micturate, the inhibition is released and the lower segmental reflex is permitted to be activated, which results in a voiding contraction of the bladder. If there is a lesion of the pyramidal system, then there will be uncontrolled or uninhibited contractions of the detrusor muscle. The contractions vary in magnitude, depending on the number of afferent stimuli activating the lower motor neurons.

11. Evans, C. L.: Physiol. Rev. **6**:358, 1926.

It is believed that the term "tone" should be confined to that condition of tension in the detrusor muscle which exists independently of connections with the central nervous system. Further increases in tension produced by impulses coming from the central nervous system should be designated as the "voiding reflex." The term "atonic bladder" should not be used to describe a bladder which cannot be emptied voluntarily but should be utilized to designate bladder muscle demonstrating a notable decrease in tension. Atonicity occurs in bladders that are permitted to distend abnormally over a varying period. Thus, distention may occur with (*a*) loss of sensation, as in patients with tabes dorsalis, (*b*) obstruction, as in patients with contracture of the vesical neck or prostatic hypertrophy or (*c*) loss of function of the entire segmental reflex arc involved in the act of micturition, as in spinal "shock." Muellner [12] has demonstrated that bladder tone is decreased during pregnancy and is on a chemical rather than on a mechanical basis.

The few observations recorded in this paper tend to suggest that the tonus of the bladder is normal during the period of spinal "shock" provided that retention of urine, with distention, is not allowed to take place.

SUMMARY

It is suggested that the muscle of the urinary bladder maintains its normal tone as well as the ability to accommodate to increasing volumes of fluid at approximately the same intravesical pressure in the condition of spinal "shock."

It is probable that the atonic bladder observed in many cases of spinal "shock" is due to retention of urine, with overdistention for a varying period, and not directly due to the loss of function of the segmental reflex arc concerned with micturition.

12. Muellner, S. R.: J. Urol. **41**:691, 1939.

TOTAL GASTRECTOMY WITH ESOPHAGODUODENAL ANASTOMOSIS

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AND
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ALTHOUGH Merrem [1] performed the first total gastrectomy in an experimental animal in 1810, Schlatter [2] in 1897 was the first to perform this operation successfully in man. Since that time it has been employed in a limited number of cases, mostly in the treatment of cancer. Because of three main reasons this operation has not been used extensively, namely, (1) the technical difficulty of its execution, (2) the high operative mortality and (3) the poor late results. At present, these factors have been overcome only partially, and therefore the procedure is still used with relative infrequency. Excellent articles dealing with total gastrectomy have been contributed by Finney and Rienhoff,[3] Pack and McNeer,[4] Graham,[5] Ransom,[6] Waugh and Fahlund,[7] Lahey [8] and others, and the subject has also been reviewed by Walters, Gray and one of us.[9]

From the Mayo Clinic and the Mayo Foundation.

Read at the fourth annual meeting of the Central Surgical Association, Chicago, Feb. 21, 1947.

1. Merrem, cited by Walters, Gray and Priestley,[9] p. 4.

2. Schlatter, C.: A Unique Case of Complete Removal of the Stomach—Successful Oesophago-Enterostomy—Recovery, M. Rec. **52**:909-914 (Dec. 25) 1897.

3. Finney, J. M. T., and Rienhoff, W. F., Jr.: Gastrectomy, Arch. Surg. **18**: 140-162 (Jan.) 1929.

4. Pack, G. T., and McNeer, G.: Total Gastrectomy for Cancer: A Collective Review of the Literature and an Original Report of Twenty Cases, Internat. Abstr. Surg. **77**:265-299, 1943; in Surg., Gynec. & Obst., October 1943.

5. Graham, R. R.: A Technique for Total Gastrectomy, Surgery **8**:257-264 (Aug.) 1940.

6. Ransom, H. K.: Total Gastrectomy, Arch. Surg., to be published.

7. Waugh, J. M., and Fahlund, G. T. R.: Total Gastrectomy, S. Clin. North America **25**:903-917 (Aug.) 1945.

8. Lahey, F. H.: Complete Removal of the Stomach for Malignancy, Surg., Gynec. & Obst. **67**:213-223 (Aug.) 1938.

9. Walters, W.; Gray, H. K., and Priestley, J. T.: Carcinoma and Other Malignant Lesions of the Stomach, Philadelphia, W. B. Saunders Company, 1942.

It is not the purpose of this paper to review the subject of total gastrectomy in general, but only to draw attention to one method of restoration of gastrointestinal continuity after complete removal of the stomach, which has received little consideration in recent years. Although esophagojejunostomy probably should remain the anastomosis of choice in the majority of cases, end to end suture of the esophagus to the duodenum can be used with certain advantages in selected cases. Esophagoduodenal anastomosis is not a new procedure, because Brigham [10] reported the first successful use of it in 1898.

According to Pack and McNeer (the table), esophagoduodenostomy was employed in 41.3 per cent of the 75 cases in which total gastrectomy was performed between the years 1884 and 1920. The mortality rate in these cases was 41.9 per cent, as compared with a mortality rate of 42.4 per cent in the cases in which an esophagojejunal anastomosis was established. In contrast, during the years 1921 to 1930 esophagoduodenostomy was performed in only 29.8 per cent of 54 cases in

Restoration of Gastrointestinal Continuity After Total Gastrectomy: Types of Anastomosis *

	Esophagojejunostomy			Esophagoduodenostomy		
Years	Cases	Percentage of Total	Mortality, per Cent	Cases	Percentage of Total	Mortality, per Cent
1884-1920	33	44.0	42.4	31	41.3	41.9
1921-1930	37	64.9	27.0	17	29.8	41.2
1931-1942	154	95.1	33.8	6	3.7	33.3

* This is an adaptation of a table of Pack and McNeer.[4]

which total gastrectomy was performed, and the mortality rate was 41.2 per cent in comparison with a rate of 27.0 per cent for the cases in which the esophagus was joined to the jejunum during the same period of years. As an obvious result, during the years 1931 to 1942 an esophagoduodenal union was established in only 3.7 per cent of the 160 cases in which the stomach was completely removed, but in this small group the mortality rate (33.3 per cent) was almost identical with that (33.8 per cent) in the cases in which the esophagus and the jejunum were united. It is apparent at once that for one reason or another esophagoduodenostomy has been employed very infrequently during the last fifteen years and that many surgeons who today have an extensive surgical practice have never employed this type of anastomosis.

Our interest in this subject was stimulated by 2 recent cases in which the pathologic process and anatomic relationships were such that

10. Brigham, C. B.: Case of Removal of the Entire Stomach for Carcinoma: Successful Esophagoduodenostomy; Recovery, Boston M & S J **138**:415-419 (May 5) 1898.

the duodenum could be readily anastomosed to the esophagus after removal of the entire stomach.

REPORT OF CASES

CASE 1.—A white man 65 years old was admitted to the hospital on Feb. 28, 1946 with a thirty-five year history suggestive of duodenal ulcer, characterized by periodic epigastric distress and repeated gastrointestinal hemorrhages. A recent hemorrhage was the cause of the patient's admission to the hospital at the time we saw him. Examination revealed that the hemorrhage was not severe and that the patient's general condition was good. The patient weighed approximately 125 pounds (56.7 Kg.). Roentgenographic examination of the stomach was reported to show an old duodenal ulcer. Gastric acidity was not studied because of the recent bleeding

After a few days of study and preparation, operation was performed on March 6, 1946. Surgical exploration revealed no demonstrable duodenal ulcer; however, high on the lesser curvature of the stomach, immediately adjacent to the esophagus, was an ulcerating lesion of indeterminate nature. Because of the definite possibility of a malignant process, the lesion was removed, and this required total gastrectomy. Since the duodenum was mobile, an end to end esophagoduodenal anastomosis was established. The pathologist reported that the specimen consisted of the entire stomach and 5 mm. of the duodenum and 5 mm. of the esophagus. There was a chronic, hemorrhagic, perforating, inflammatory ulcer, with a crater 2 cm. in diameter, situated at the esophagogastric juncture.

The postoperative course of the patient, to the time of this writing, has been satisfactory. He was dismissed from the hospital on the fourteenth postoperative day. He returned to work several weeks later and has been working daily since that time. He weighed 20 pounds (9.1 Kg.) less than his normal figure when he was discharged from the hospital. When he was examined eight months postoperatively he had gained 10 pounds (4.5 Kg.) and the value for hemoglobin was normal.

CASE 2.—A woman 66 years old registered on March 27, 1946 because of loss of weight (38 pounds or 17.2 Kg.) during the previous two months and the more recent development of nausea and vomiting. Examination revealed a rather weak woman who weighed only 110 pounds (49.9 Kg.). Roentgenographic investigation revealed an obstructing lesion in the region of the pylorus. The stomach still contained barium which had been administered five days previously

After adequate preoperative preparation, operation was performed on April 3, 1946, at which time total gastrectomy, with removal of the omentum, and end to end esophagoduodenostomy were performed. A linitis plastica type of carcinoma was seen to involve virtually the entire stomach. The pathologist reported that the lesion was a grade 4 scirrhous adenocarcinoma with multiple portions of mucosal ulceration and involvement of the submucosa of the attached portions of the duodenum and esophagus. There was also invasion by the malignant process of the serosa of the stomach, the regional lymph nodes and the omentum.

The early postoperative course was complicated by atelectasis; the patient did not leave the hospital until the twenty-third postoperative day. She was readmitted four days later because of empyema which required open drainage. Her condition improved gradually, and she was dismissed eighteen days later. There was no indication that the type of esophageal anastomosis established was in any way associated with the pulmonary complications. The patient succumbed six months later, from the malignant process.

SELECTION OF PATIENTS

Obviously, it is not feasible to bring the ends of the esophagus and the duodenum together without tension and with an adequate blood supply to both parts in every case in which total gastrectomy is performed. In fact, no attempt should be made to establish such a union in all cases. However, in the thin, asthenic type of person, whose costal margins form an acute angle at the xiphoid process and whose viscera frequently are more mobile than those found in the average patient, the duodenum can be joined to the end of the esophagus, easily and with no tension. It is in this type of patient that establishment of such an anastomosis is suggested and recommended. In one of the cases reported it was unnecessary to mobilize the duodenum in any way. In the other case the lateral peritoneal attachment of the duodenum was incised for a distance of a few centimeters. This can be done without interference with the blood supply of the duodenum. We do not know the exact percentage of cases in which such an anastomosis is practical, but if it is kept in mind as a possibility we believe that it will be found applicable in a fair percentage of cases. Perhaps our interest in this subject has been stimulated by our more widespread use of the Billroth I type of anastomosis, with the Schoemaker modification, during the last several years, in the treatment of both duodenal and gastric ulcers as well as in selected cases of small malignant lesions in the region of the pylorus. Usually, the thick-chested, heavy-set person, who commonly also has a short neck, is not a suitable candidate for either esophagoduodenostomy or a Billroth I type of operation. Decision as to the establishment of esophagoduodenal anastomosis, in general, must be deferred until the abdomen has been opened and the situation thoroughly surveyed.

TECHNIC

The technical execution of total gastrectomy with esophagoduodenal anastomosis is not difficult when it is performed for a properly selected patient. In brief, after the abdomen has been opened and it has been decided that total gastrectomy is advisable, the surgeon proceeds with mobilization of the stomach in the usual manner (fig. 1). The duodenum is severed immediately distal to the pylorus, if there is no pathologic process present in this area. The duodenal stump is held in a curved forceps or similar type of clamp and is not closed unless it is apparent or becomes apparent that union of the esophagus and the duodenum cannot be accomplished without tension. The surgeon then continues to free the stomach until it is attached only to the esophagus. The stomach should be emptied by suction or a clamp should be placed across the proximal portion of the cardia before the stomach is reflected upward and outward at the cephalad end of the incision, in order to prevent regurgitation of gastric contents into the esophagus. Then the end of the duodenum is approximated to the esophagus to determine whether it will lie in this position freely and without the exertion of traction on its attachments.

If such is not the case, the peritoneum lateral to the duodenum may be incised for a few centimeters, and gentle mobilization of the duodenum with no interference with its blood supply may be accomplished. If the duodenal stump still does not

lie freely in approximation with the esophagus, this type of anastomosis should be abandoned, the duodenal stump should be closed and esophagojejunostomy should be performed.

If it is found that the duodenum will lie in contact with the esophagus when all traction is removed, the surgeon may proceed with anastomosis of these two structures. First, two or three, preferably three, interrupted stitches of silk are placed between the posterior aspect of the duodenum and the pancreas (fig. 2*a*). These

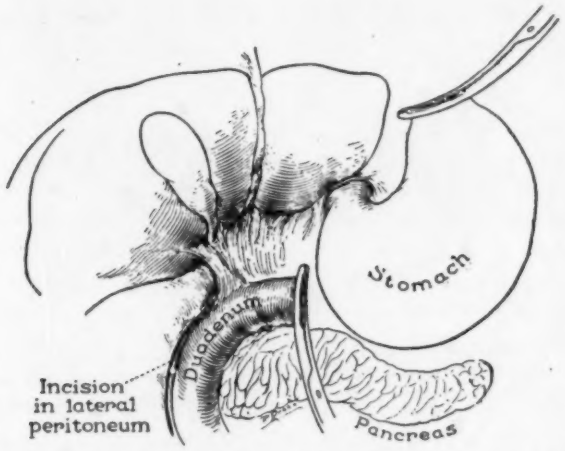

Fig. 1.—First step in performance of total gastrectomy and establishment of esophagoduodenal anastomosis. The stomach is mobilized completely except for its attachment to the esophagus. The duodenal stump is held in an appropriate type of clamp. If necessary, the duodenum is mobilized by an incision in its lateral peritoneal attachment.

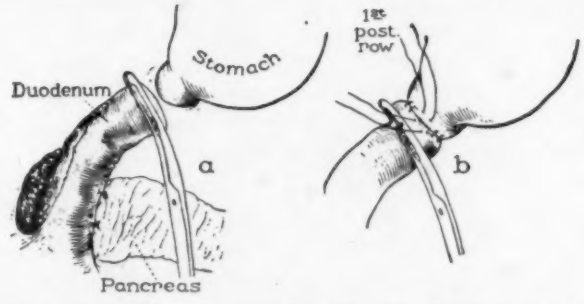

Fig. 2.—Esophagoduodenostomy (continued): (*a*) after it has been determined that the duodenum can be approximated to the esophagus without tension, the duodenum is anchored to the pancreas with approximately three interrupted stitches; (*b*) the first posterior row of interrupted stitches of silk is placed between the duodenum and the esophagus.

stitches, which constitute no part of the anastomosis and are removed at least 3 or 4 cm. from the junction of the esophagus and the duodenum, serve to fix the duodenum in its approximation to the esophagus. We prefer a three row suture technic which consists of the placing of an outer row of interrupted stitches of silk (fig. 2*b*), followed by a continuous row of silk (fig. 3*a*) and, finally, a

row of surgical gut in the mucosa (fig. 3 *b*). The posterior rows are placed first, from without in, so that the mucosal row is last of the posterior suture lines to be completed. The esophagus is left intact until the posterior mucosal row is started, and then an incision is made transversely across the posterior wall of the esophagus (fig. 3 *b*). At this time the esophagus is emptied by suction, if it has any contents. Next, the clamp is removed from the duodenum and the crushed tissue is

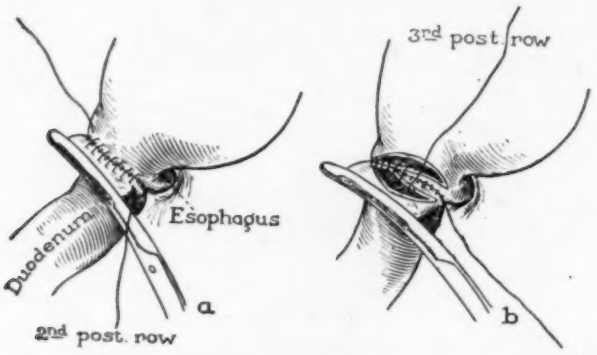

Fig. 3.—Esophagoduodenostomy (continued): (*a*) a second posterior row of continuous silk suture is completed; (*b*) the third and innermost posterior row of suture of chromic surgical gut is placed in the mucosa of the duodenum and esophagus.

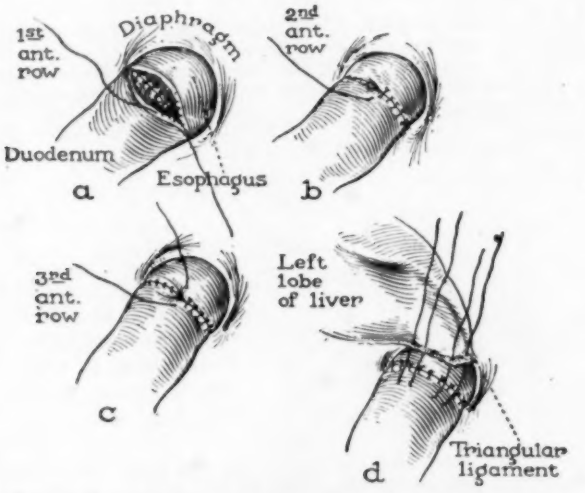

Fig. 4.—Esophagoduodenostomy (continued): (*a*, *b* and *c*) three rows of sutures are placed anteriorly to correspond with those placed posteriorly; (*d*) the edge of the left triangular hepatic ligament is sutured to the duodenum, several centimeters below the completed anastomosis.

trimmed away. After the posterior mucosal row of sutures has been completed, the anterior wall of the esophagus is incised gradually as the first anterior mucosal suture line is completed (fig. 4 *a*). The outer two rows of sutures then are placed anteriorly (fig. 4 *b* and *c*). After completion of the anastomosis, the duodenum is

anchored in its new position anteriorly, just as it was anchored to the pancreas posteriorly. This may be done in one of two ways, depending on the convenience afforded by the local anatomic relationships. Usually, the left triangular ligament which attaches the edge of the left lobe of the liver to the diaphragm has been severed to aid in mobilization and visualization of the esophagus. The severed edge of this ligament can be brought down over the anastomosis and sutured to the duodenum several centimeters below the anastomosis (fig. 4*d*). If this ligament is not approximated readily to the duodenum, the latter can be fixed with a few sutures to the under surface of the left lobe of the liver.

COMMENT

It appears that anastomosis of the esophagus to the duodenum has been a rather forgotten procedure during the past fifteen years or so, and this without adequate justification. If the data collected by Pack and McNeer are referred to and the mortality rates after both esophagoduodenostomy and esophagojejunostomy are examined, it is seen that for all years except 1921 to 1930 the rates are comparable. During this one ten year period the mortality rates favor esophagojejunal anastomosis; however, only 17 cases of esophagoduodenal anastomosis were reported during this time, a number far too small to be statistically significant. Thus, the argument that a higher mortality rate is associated with this procedure hardly seems valid. When esophagoduodenal anastomosis is used indiscriminately, it is understandable that the risk might be greater, but if it is employed only in selected cases in which a well established anastomosis can be accomplished without tension, there is little if any apparent reason why the risk should not be as low as that associated with any other procedure. In fact, there are some reasons to suspect that the mortality rate might be lower. In the first place, when it is performed under proper circumstances, the operation is easier of execution than esophagojejunostomy because there is only one loop of intestine to deal with, and this can be readily approximated to the esophagus. Moreover, there are fewer suture lines, because the stump of the duodenum is not closed. The questions of performance of enteroanastomosis and anterior or posterior colic types of anastomosis do not even arise for consideration.

In all types of surgery one principle which seems uniformly significant, even though the reasons for its importance may not always be apparent, is that the closer to normal the surgeon can leave the anatomic arrangement of the patient, other things being equal, the more nearly physiologically ideal and desirable it seems. Thus, if the stomach is to be removed completely, the normal is approximated more closely if food enters the duodenum, rather than the jejunum, as it leaves the esophagus. In this manner it comes into direct contact with the digestive juices of the duodenum and follows a normal pathway from there on. It is obvious that our experience with this procedure has been too limited to permit dogmatic statements and conclusions to be formed.

It has seemed warranted, however, that mention be made of certain technical advantages, as well as a few perhaps theoretic advantages, which this procedure possesses.

SUMMARY

Previous experience with esophagoduodenal anastomosis after total gastrectomy has been mentioned briefly. Two cases in which this type of procedure recently was performed have been presented. It was stressed that this procedure is suitable only in a selected group of patients. Technic for the operation has been briefly described. Esophagoduodenostomy is considered advantageous when practical because of its comparative simplicity and elimination of unnecessary suture lines and also because of the more nearly normal anatomic and physiologic restoration of gastrointestinal continuity which results.

LIGATION OF THE SPLENIC ARTERY IN PATIENTS WITH PORTAL HYPERTENSION

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AND
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WITH the improvement and refinement of the technic of portacaval anastomosis by Whipple [1] and Blakemore and Lord,[2] an important addition was made to the armamentarium of the surgeon in his approach to the difficult problem of portal hypertension. In the past year Blakemore [3] has reported 14 cases of portacaval shunt performed for the alleviation of portal hypertension. In this series there were 2 operative deaths. One patient died one year postoperatively, and another succumbed two years and three months after operation. Although the follow-up of the surviving patients was of necessity short, this operative procedure appears to offer the greatest promise at the present time for relief of the distressing effects of portal hypertension.

It is, however, an operation of considerable magnitude. It requires hours for its satisfactory completion and places a tremendous additional load on an already overtaxed patient. Because of these facts, there will be some patients with portal hypertension who could not be subjected to a portacaval shunt type of operation without the occurrence of a prohibitive operative mortality rate. For these patients a less trying surgical procedure must be found. Although admittedly the procedure of less magnitude will give less improvement, yet in the light of a comparison of operative mortality rates it may appear as the more rational approach to the problem in treatment of patients who are poor risks.

Read at the fourth annual meeting of the Central Surgical Association, Chicago, Feb. 22, 1947.

From the Department of Surgery, University of Illinois College of Medicine, and the Illinois Research and Educational Hospitals.

1. Whipple, A. O.: The Problem of Portal Hypertension in Relation to the Hepatosplenopathies, Ann. Surg. **122**:449, 1945.

2. Blakemore, A. H., and Lord, J. W., Jr.: The Technic of Using Vitallium Tubes in Establishing Portacaval Shunts for Portal Hypertension, Ann. Surg. **122**:476, 1945.

3. Blakemore, A. H.: Portacaval Anastomosis: A Report on Fourteen Cases, Bull. New York Acad. Med. **22**:254, 1946.

Prior to the performance of portacaval anastomosis, the surgical procedures most commonly used in the treatment of portal hypertension were omentopexy or splenectomy.

The first successful omentopexy was performed in 1895 by Morison,[4] whose patient lived for two years after operation. However, Talma [5] is given the credit for popularizing the procedure. Cates [6] in 1943 compared 117 consecutive cases of cirrhosis in which the patients were subjected to purely medical management with 38 cases in which omentopexy was performed. He found that 72 per cent of the patients treated nonoperatively died within one year and that 68 per cent of the patients subjected to omentopexy died within the same period. He consequently concluded that omentopexy was no more satisfactory than medical management.

It is known that the spleen furnishes approximately 40 per cent of the blood to the portal circulation. In the presence of splenomegaly the contribution is no doubt greater. Consequently, splenectomy has been performed for some time in the treatment of portal hypertension; this procedure does frequently afford some improvement, for a time at least, until the portal pressure builds up to the hypertensive level present before the operation. However, in the presence of portal hypertension splenectomy is in itself an operation of no little magnitude, and among patients who are poor risks the mortality rate will be prohibitive considering the fact that the benefit is only temporary. In a patient for whom the risk is slight or at least average the portacaval shunt type of procedure is preferable.

The procedure which we wish to emphasize for use in patients with portal hypertension who are poor risks is that of ligation of the splenic artery. Ligation of the splenic artery is followed by gradual atrophy of the spleen. Thus, in effect, stoppage of the arterial supply to the spleen results in a functional splenectomy. It is a procedure which can be accomplished in much less than an hour. This operation is a relatively minor surgical procedure when compared with splenectomy or portacaval shunt, and we believe that the results will warrant its consideration.

As a matter of fact, the results of ligation of the splenic artery should be superior to those of splenectomy because the veins transporting blood from the spleen are undisturbed and thus channels of escape for the portal blood are left. The splenic vein normally receives tributaries from the short gastric veins, the left gastroepiploic vein and the pancreatic

4. Drummond, D., and Morison, R.: A Case of Ascites Due to Cirrhosis of Liver Cured by Operation, Brit. M. J. **2**:728, 1896.

5. Talma, S.: Chirurgische Öffnung neuer Seitenbahnen für das Blut der Vena porta, Berl. klin. Wchnschr. **35**:833, 1898.

6. Cates, H. B.: Surgical Treatment for Cirrhosis: Prognosis Subsequent to Omentopexy, Arch. Int. Med. **71**:183 (Feb.) 1943.

veins. These veins become markedly dilated, and on certain occasions the blood flow is no doubt reversed. For example, blood may flow from the splenic vein into the pancreatic veins and then posteriorly into the systemic system; likewise, blood may flow from the splenic vein into the short gastric or gastroepiploic veins, superiorly into the esophageal veins or into the veins in the diaphragmatic attachments. In portal hypertension dilated veins form also in the phrenicolienal ligament, through which blood may flow posteriorly into the systemic circulation. In addition to these outlets, large veins develop in adhesions between the spleen and the parietal peritoneum. Therefore, it is conceivable that with the splenic artery tied and the spleen itself undisturbed there would be a better opportunity for escape of portal blood than with the spleen removed. We have ligated the artery in 3 patients and we have encountered no evidence of necrosis of the spleen although there was a sharp reduction in the size.

The technic of ligation of the splenic artery is simple and can be described briefly. Through an incision in the left upper part of the abdomen the vessel can be isolated in either of two ways. By incision of the gastrohepatic omentum it can be secured just distal to its origin from the celiac axis. However, we prefer to isolate the vessel by opening into the lesser peritoneal cavity through an incision made in the gastrocolic omentum. This incision will reveal the hilus of the spleen and the tail of the pancreas. About 6 cm. from the hilus of the spleen and just at the superior edge of the pancreas the splenic artery projects upward so that it is either plainly visible in the field or can be isolated by blunt dissection.

Palpation with the index finger may be of aid in locating the vessel if it is not readily visible; dissection is best carried out with a blunt-nosed instrument, such as a Péan or a Mixter forceps, since the vein often lies close to the artery at this point. In patients with a large spleen the artery will likewise be large and with few exceptions will be plainly visible because of its tortuosity. Because the artery is commonly calcified and friable in elderly persons with many types of portal hypertension, coarse suture material should be used. Since vessels ligated in continuity commonly recanalize, it is probably advisable to ligate the vessel and cut between the ligatures. To assure safety of ligation, two ligatures should be applied on the proximal stump. After ligation of the vessel, the opening in the gastrocolic omentum should be closed with several interrupted sutures of fine cotton or a continuous suture of size 0000 or 00000 surgical gut.

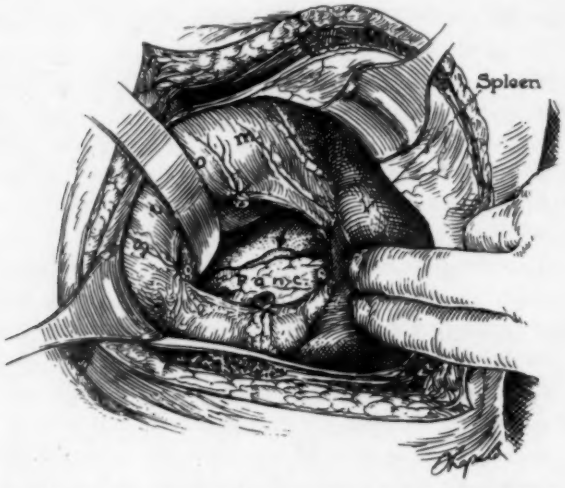
Blain [7] in 1918 first reported ligation of the splenic artery in a patient with Banti's disease. The patient was a 38 year old white man with

7. Blain, A. W.: Ligation of Splenic Artery, Surg., Gynec. & Obst. **26**:660, 1918.

known splenomegaly for nineteen years. No ascites or hematemesis was present. At the time of the report (four and one-half years after operation) the patient was entirely well and doing heavy work.

Watson [8] reported good results from ligation of the splenic artery in a 26 year old white woman with known splenic enlargement since the age of 7. This patient had had several severe attacks of hematemesis and considerable ascites preoperatively. After ligation of the artery, abdominal paracentesis was still necessary but less frequently. There were no episodes of hematemesis three years postoperatively, at which time the patient was in good health and able to work.

Berg and Rosenthal [9] reported 3 cases of cirrhosis of the liver and splenomegaly in which the treatment was ligation of the splenic artery.



Ligation of the splenic artery at the superior edge of the pancreas, approached by incising through the gastrocolic omentum. Arrow indicates point of ligation. (Modification of Cole's procedure, figure 8 in *Surg. Clin. North America* **22**:46 [Feb.] 1942).

All the patients had a preoperative history of hematemesis. Two of the patients died within a year of the operation. (One died postoperatively after a splenectomy performed ten months after ligation of the artery.) The third patient died three years postoperatively from an exanguinating hemorrhage of the gastrointestinal tract. This patient had felt markedly improved during the three years after operation.

8. Watson, R. B.: Ligation of Splenic Artery for Advanced Splenic Anaemia, Brit. M. J. **1**:821, 1935.

9. Berg, A. A., and Rosenthal, N.: Ligation of the Splenic Artery for Thrombocytopenic Purpura and Congestive Splenomegaly, J. Mt. Sinai Hosp. **8**:382, 1942.

Maggiore [10] and Alessandri [11] have reported good results with ligation of the splenic artery for treatment of congestive splenomegaly.

As indicated previously, the procedure is by no means new, but it has been used comparatively little considering the fact that the few reports available in the literature are in favor of its use as a palliative procedure.

Our experience with ligation of the splenic artery in the treatment of patients with portal hypertension who are poor risks is reported in the following cases:

REPORT OF CASES

CASE 1.—*History.*—T. S., a white man aged 62, was admitted to the Research and Educational Hospitals on Jan. 15, 1946. He had been well until May 1945, when he noted sudden and almost simultaneous appearance of a hernia in the right side of the scrotum, swelling of the abdomen and an umbilical hernia. He dated his difficulties back to November 1944, when he strained himself to prevent a 200 pound (90 Kg.) weight falling on him. The patient was given mersalyl by a physician in May 1945, and there was some decrease in abdominal swelling after the administration of the drug. This was repeated four or five times weekly, with varying results. Two abdominal tappings were performed at a private hospital, and the patient obtained temporary relief. There was no history of the consumption of alcohol in the three years preceding his admission to the hospital. Prior to that time he drank only one glass of wine or beer daily. There was no history of hematemesis or melena.

Physical Examination.—The blood pressure was 120 systolic and 80 diastolic, the pulse rate 84, the respirations 18 and the temperature 99 F. Physical examination revealed a well developed, well nourished, white man who did not appear acutely ill. The abdomen was extremely distended, and a fluid wave and shifting dulness were present. The liver was palpable 5 fingerbreadths below the costal margin. The spleen was palpable 4 fingerbreadths below the costal margin. A large umbilical hernia and one in the right inguinal area were present. The findings in the remainder of the physical examination were essentially normal.

Laboratory Data.—Examination of the blood revealed a red blood cell count of 4,890,000, a hemoglobin content of 10.5 Gm. per hundred cubic centimeters and a white blood cell count of 4,500. The total serum protein was 7.3 Gm. per hundred cubic centimeters, with albumin 4.5 Gm. and globulin 2.8 Gm. The icterus index was 6. The blood glucose level was 86 mg. and the plasma chloride content (as NaCl) 525 mg. per hundred cubic centimeters. The nonprotein nitrogen was 43 mg. per hundred cubic centimeters. The reaction to a Wassermann test was negative. The sulfobromophthalein test showed no retention of dye in forty-five minutes.

Operation.—With the patient under cyclopropane anesthesia an abdominal operation was performed on Jan. 30, 1946. The liver was found to be considerably enlarged and was indurated with a finely nodular surface, confirming the diagnosis of cirrhosis of the liver. The spleen was enlarged almost to the size of a man's head. There were no adhesions on its surface. Portal hypertension was obvious since

10. della Maggiore, B. D.: Effeto favorevole e duraturo della allacciatura dell'arteria splenica in sindrome spleno-epatica ascitogena, Policlinico **48**:56, 1941.

11. Alessandri, R.: Experiences with Surgery of the Spleen: Report of Two Unusual Cases, J. Mt. Sinai Hosp. **4**:489, 1938.

bleeding from tiny venules was profuse. The splenic artery was ligated at the superior border of the pancreas. The spleen contracted markedly after ligation of the artery. An omentopexy was performed.

Postoperative Course.—The immediate postoperative course was uneventful, and the patient was discharged from the hospital on Feb. 10, 1946. No ascites was present at this time.

On March 4 the patient was noted to have a moderate amount of fluid in the abdomen but not sufficient to require tapping. The spleen was barely palpable at this time.

On June 17 the abdomen was considerably distended with fluid. Intake of fluid was restricted, and the patient was given 15 grains (0.97 Gm.) of ammonium chloride four times a day. By July 24 practically all the abdominal fluid had disappeared. The spleen at this date was palpable two and one-half fingerbreadths below the costal margin.

On Jan. 8, 1947, no abdominal fluid was present. The spleen was palpable one and one-half fingerbreadths below the costal margin. The patient had a hernia half the size of a grapefruit at the site of the omentopexy. He has felt much stronger since the operation and is now working.

CASE 2.—*History.*—P. M., a white man aged 44, was admitted to the Research and Educational Hospitals on March 7, 1944, having been transferred from a state mental hospital. He had been institutionalized since 1927 for treatment of dementia precox and was first hospitalized on Dec. 24, 1943, because of pronounced abdominal distention. A physical examination at this time had revealed a spleen palpable 3 fingerbreadths below the costal margin. A paracentesis was performed on Dec. 26, 1944, and 9,000 cc. of clear, straw-colored fluid with a specific gravity of 1.010 was withdrawn. The ascites recurred within two weeks and then remained stationary. No further paracenteses were done. There was no history of alcoholism or of hematemesis or melena.

Physical Examination.—The blood pressure was 154 systolic and 98 diastolic, the pulse rate 86, the respirations 18 and the temperature 98.2 F. Physical examination revealed an emaciated, chronically ill, white man who appeared mentally incompetent and childish. There was a slight icteric tinge to the conjunctivas. The abdomen was markedly distended, and a fluid wave and shifting dulness were present. The bowel sounds were faint. No masses or abdominal organs were palpable. The abdominal veins were not prominent. The findings in the remainder of the physical examination were essentially normal.

Peritoneoscopy was performed on March 11, 1944, and approximately 500 cc. of straw-colored fluid was aspirated. The liver was examined under direct vision and seen to be small and grayish tan, with multiple nodules resembling mulberries. The spleen could not be visualized.

Laboratory Data.—Examination of the blood revealed a red blood cell count of 4,990,000, a hemoglobin content of 65 per cent and a white blood cell count of 10,400. The total serum protein content was 7.3 Gm. per hundred cubic centimeters, with albumin 3.5 Gm. and globulin 3.8 Gm. The icterus index was 10. The blood glucose level was 96 mg. and the plasma chloride content (as NaCl) 493 mg. per hundred cubic centimeters. The nonprotein nitrogen was 29 mg. per hundred cubic centimeters of blood. Reactions to the Kahn and Kolmer tests were negative. The results of the tests of hepatic function were as follows: There was a 4 plus reaction to the test for cephalin-cholesterol flocculation, the sulfobromophthalein test showed 10 per cent retention of dye in five minutes and 2.5 per cent retention in thirty minutes and in the hippuric acid test 52 per cent was excreted. The venous pressure was 165 mm. of water.

Operation.—With the patient under cyclopropane-ether anesthesia, an abdominal operation was performed on March 29, 1944. The liver was found to exhibit typical Laennec's cirrhosis. The spleen was enormously enlarged and extended almost to the crest of the ilium. The patient's poor physical condition scarcely appeared to justify a portacaval shunt. It was therefore decided to ligate the splenic artery. An opening was made in the gastrohepatic ligament, but the celiac axis was surrounded by so much edematous tissue that it was decided to isolate the artery to the left of the stomach in the lesser peritoneal cavity. The artery was isolated at the superior margin of the pancreas and tied with size 1 chromic surgical gut. An omentopexy was performed.

Postoperative Course.—The patient's abdomen slowly became distended with fluid, and on April 13, 8,000 cc. of straw-colored fluid was withdrawn by abdominal paracentesis. Since discharge from the hospital on April 13, 1944, he has required frequent tapping of the abdomen for removal of ascitic fluid. In addition, the patient now has a massive ventral hernia at the site of the omentopexy. However, he has been subjectively more comfortable postoperatively than he was prior to surgical intervention.

CASE 3.—*History.*—J. L., a white man aged 40, was admitted to the Research and Educational Hospitals on Nov. 22, 1943, having been transferred from a state mental hospital. When he was first admitted to the mental hospital in January 1942 he was found to have ascites. This disappeared spontaneously. The patient did not then note an increase in the size of his abdomen until September 1943. Abdominal paracenteses for withdrawal of ascitic fluid were performed and the following amounts withdrawn: Sept. 11, 1943, 9,000 cc.; October 23, 15,000 cc.; November 8, 17,500 cc., and November 21, 15,800 cc. There was a history of previous alcoholism but none since 1942. In 1939 the patient felt ill one morning and vomited a quart (946 cc.) of red-tinged gastric fluid. Tarry stools were noted at this time. There were no similar episodes.

Physical Examination.—The blood pressure was 110 systolic and 60 diastolic, the pulse rate 86, the respirations 18 and the temperature 98.2 F. The abdomen was notably distended, and a definite fluid wave was present. The liver, kidneys and spleen were not palpable. The abdominal veins were prominent. The findings in the remainder of the physical examination were essentially normal.

Laboratory Data.—Examination of the blood revealed a red blood cell count of 4,400,000, a hemoglobin content of 12.0 Gm. per hundred cubic centimeters and a white blood cell count of 3,400. The total serum protein content was 5.2 Gm. per hundred cubic centimeters, with albumin 3.1 Gm. and globulin 2.1 Gm. The blood glucose level was 98 mg. per hundred cubic centimeters and the plasma chloride content (as NaCl) 521 mg. The nonprotein nitrogen was 30.3 mg. per hundred cubic centimeters of blood. The reactions to the Wassermann and Kahn tests were negative. The results of tests of hepatic function were as follows: The cephalin-cholesterol flocculation test gave a 3 plus reaction and the sulfobromophthalein test showed 40 per cent retention of dye in five minutes and 30 per cent retention in thirty minutes. The venous pressure was 60 mm. of water.

Operation.—An abdominal paracentesis was performed as a preliminary procedure on Nov. 29, 1943, with the withdrawal of 16,000 cc. of straw-colored fluid.

With the patient under cyclopropane-ether anesthesia, an abdominal operation was performed on December 2. The liver appeared normal in size and shape, with at most a slight increase in fibrous tissue. The spleen was six to eight times its normal size and was covered over its entire extent with adhesions, which

bound it firmly in its bed. The adhesions were old and fibrosed and contained large numbers of vessels, some up to 2 or 3 mm. in diameter. It was felt that the pathologic changes were primarily those of thrombosis of a splenic vein and that the patient would not tolerate a splenectomy because of the massive adhesions and poor physical condition. Consequently, the splenic artery was ligated above the middle of the pancreas, and an omentopexy was performed.

Postoperative Course.—The immediate postoperative course was uneventful. The patient was discharged from the hospital on Dec. 16, 1943. At this time his abdomen was moderately distended with fluid, but it was not tight. After discharge from the hospital he required repeated abdominal tapping, with withdrawal of large amounts of ascitic fluid. On Feb. 12, 1944, he had a severe hematemesis, and he died on March 11. Permission for autopsy was not granted.

SUMMARY

After ligation of the splenic artery 1 of our patients showed immediate improvement, which has persisted up to date (one year after operation). We interpret the result in this case as excellent, although the patient must be followed for several years more before the final answer can be given. The second patient is still alive, almost three years since operation. Ascites has not improved appreciably. However, the patient has noted subjective improvement. This, together with the length of the survival period, leads us to believe that the ligation of the splenic artery was of some benefit. The third patient died four months after ligation.

In spite of the fact that in the 3 cases herein reported we performed an omentopexy in addition to ligation of the splenic artery, our experience with omentopexy and the work of Cates [6] lead us to believe that the favorable results are not due to this procedure.

Although ligation of the splenic artery will not and should not replace portacaval shunt in the treatment of portal hypertension, we believe that it is definitely indicated in patients who could not tolerate the more extensive procedure. Even though there is an appreciable decrease in the size of the spleen after ligation, we have observed no evidence of necrosis or deleterious effects attributable to the rapid atrophy or necrosis. It is possible that ligation of the splenic artery in patients without portal hypertension might lead to undesirable or serious effects from rapid necrosis; we have not performed ligation of the artery in such circumstances.

AEROEMBOLISM OF BONE MARROW

An Experimental Study

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THE CLINICAL recognition of aseptic necrosis of bone is based largely on the appearance of changes in the roentgenogram, and these of necessity must be the result of slowly developing factors on one of the resisting tissues of the body—bone. Death of bone tissue without evidence of infection has been described in various regions of the body in both childhood and adult life, and the factor or factors affecting its prevention or progress are of practical value. The bone infarct in the diaphysis may be asymptomatic, but whenever the pathologic process involves one of the articular surfaces, it will give rise to limitation in the joints and produce severe disability. In childhood Freiberg's disease, Perthes' disease and Köhler's disease are all representative of a localized aseptic necrosis. In the adult the development of aseptic necrosis in the head of the femur following a traumatic dislocation or a fracture of the neck of the femur presents an extremely serious problem.

This experimental study was begun about a year ago. We believe that we have demonstrated some of the early changes in the bone that precede the roentgenographic evidence, but this report is simply a preliminary one and represents an incomplete investigation to demonstrate one of the many ways in which aseptic necrosis may possibly be produced. In children, with their ability for rapid repair, complete revascularization of aseptic areas is frequently observed. In adults, while "creeping substitution" does occasionally cause a revascularization of an aseptic necrotic area, this occurrence is the exception rather than the rule.

A primary factor in the production of aseptic necrosis of bone is disruption of the blood supply, with loss of nutrition to the part of the bone involved. The different processes by which the bone is

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deprived of its blood supply and nutrition, with development of aseptic necrosis, are still a subject of speculation.

Moritz [1] among others has pointed out that trauma plays an important role. However, the evidence implicating trauma is largely clinical, an example being the development of osteochondritis dissecans in the knees and elbows of athletes. Hirsch [2] regarded mechanical irritation as the factor leading to an obliterating endarteritis of the nutrient vessels and reported that in 9 of the 10 cases studied by him satisfactory histologic evidence of vascular obliteration was revealed. It has been suggested that prolonged trauma may lead to a vasomotor disturbance of sufficient intensity and duration to initiate ischemia and aseptic necrosis. Axhausen in 1922 reported that aseptic necrosis is the result of embolic infarction.

Circulatory occlusion, therefore, in one form or another is the primary basis for the production of aseptic necrosis.

EXPERIMENTAL WORK

Phemister [3] has stated that since the studies of Langer in 1875 the bones have been known to contain end arteries, yet knowledge of the changes following vascular occlusion has developed slowly. The textbooks on pathology rarely mention bone infarcts and aseptic necrosis, and the experimental work on this subject is meager.

According to the earliest record which we have been able to find, the experimental production of aseptic necrosis began when Bancroft [4] injected irritating substances into the medullary cavity of the bone. In 1922 he used croton oil. Other investigators used other chemical irritants. However, the results imitated infection rather than aseptic necrosis as it is seen clinically and usually led to sequestration of bone, with evidence of inflammation of soft tissue.

Later experimenters began placing various foreign bodies in the arteries to produce embolism and infarction. Kistler [5] produced these by injection of a 2 per cent solution of charcoal in isotonic solution of sodium chloride containing 5 per cent acacin into the nutrient artery of rabbits. Another type of aseptic emboli has been produced by placing tiny glass beads in the circulation to cause infarction.

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1. Moritz, A. R.: Pathology of Trauma, Philadelphia, Lea & Febiger, 1942, pp. 350-353.

2. Hirsch, E. F.: Arterial Occlusion with Aseptic Necrosis of Bone, Arch. Surg. **37**:926-943 (Dec.) 1938.

3. Phemister, D. B.: Changes in Bones and Joints Resulting from Interruption of the Circulation: General Consideration and Changes Resulting from Injuries, Arch. Surg. **41**:436-472 (Aug.) 1940.

4. Bancroft, F. W.: Bone Repair Following Injury and Infection, Arch. Surg. **5**:646-677 (Nov.) 1922.

5. Kistler, G.: Experimental Infarction of the Femur in Rabbits, Arch. Surg. **29**:589-611 (Oct.) 1934.

Johnson,[6] in a series of experiments in 1927 designed primarily to study the circulation of bone, produced aseptic necrosis. He stripped the periosteum from the femur of dogs, and after tying off the nutrient artery he plugged the medullary canal with wax.

Brunschwig [7] in 1930 found that stripping the periosteum from the bones of young dogs resulted in the production of aseptic necrosis.

ASEPTIC NECROSIS OF BONE IN CAISSON DISEASE

One of the best examples of widespread aseptic necrosis of bone occurs in caisson disease, which has been observed for many years. Men have worked under increased pressure since the sixteenth century. Paul Bert,[8] a Frenchman, first published some of the factors which caused caisson disease in a series of experiments in 1878. It has since been substantiated that this clinical picture is a direct result of nitrogen bubbles forming in the tissues and in the circulation after rapid decompression of compressed animals.

When an animal is subjected to compression with air, the fats and lipids have the ability to absorb nitrogen five to six times as rapidly as the nonfatty tissues. Air is composed of about 20.9 per cent oxygen, 78 per cent nitrogen and 0.04 per cent carbon dioxide. Large fatty deposits are in the abdomen, the central nervous system and the long bones. It has been estimated that in man there is complete saturation of the tissues with nitrogen in five hours. After sudden decompression the nitrogen is released from the circulation in much greater quantities than can be adequately absorbed, and this results in stasis of the released nitrogen, with the formation of nitrogen bubbles in the tissues and blood. These bubbles may cause circulatory or respiratory collapse, injuries of the central nervous system and lesions in the bones and other tissues. Meakins [9] stated: "If the decompression is not done properly bubbles of nitrogen will form in the tissues, capillaries and small veins and venules. These will be particularly prominent in the fat deposits, the white matter of the central nervous system of the brain and nerves and other tissues where the saturation is high or *the blood flow is slow.*" The italics are ours, but this point is worth consideration.

In recent years many articles have appeared in the literature describing lesions of the bone in caisson workers. Kahlstrom, Burton and

6. Johnson, R. W., Jr.: A Physiological Study of the Blood Supply of the Diaphysis, J. Bone & Joint Surg. **9**:153-184, 1927.

7. Brunschwig, A.: Experimental Infarction of Bone Marrow, Proc. Soc. Exper. Biol. & Med. **27**:1049-1051, 1930.

8. Bert, P.: La pression barométrique Paris, G. Masson, 1880.

9. Meakins, J. C.: Practice of Medicine, ed. 2, St. Louis, C. V. Mosby Company, 1938.

Phemister [10] have described infarction of bone in caisson disease resulting in encapsulated and calcific areas in the diaphysis. Herzmark,[11] James,[12] Taylor,[13] Bell, Edson and Hornick,[14] Rendich and Harrington,[15] Walker [16] and others all described lesions of the bone in caisson workers. These various reports can be summarized as follows. First, the lesions were in the main confined to the metaphyses, although both diaphysial and articular lesions have been reported. Second, the lesions were all of long duration. Early lesions in bone have not been described. Third, the lesions were all thought to be the result of absorption by air emboli in the vessel or of pressure of nitrogen gas bubbles on the vessels from without or of a combination of these leading to the development of aseptic necrosis. On the other hand, many examples can be

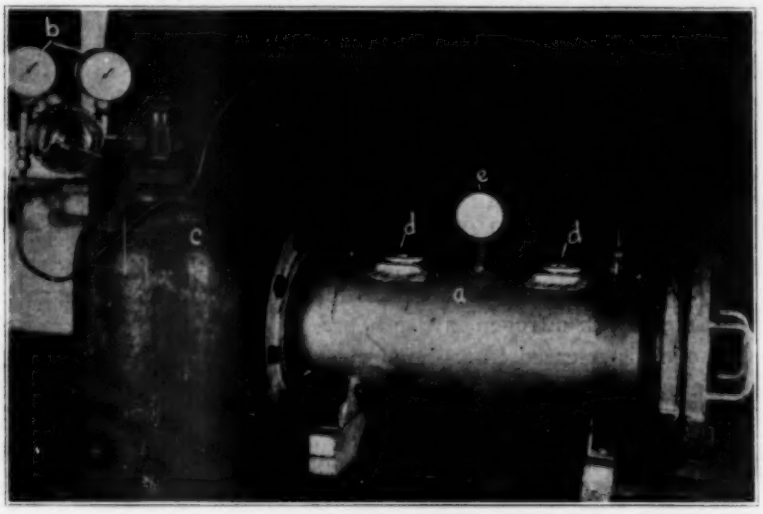

Fig. 1.—Type of compression chamber used, showing (*a*) pressure chamber, (*b*) pressure gages, (*c*) compressed air tank, (*d*) observation windows and (*e*) check pressure gages.

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10. Kahlstrom, S. C.; Burton, C. C., and Phemister, D. B.: Infarction of Bones in Caisson Disease Resulting in Encapsulated Areas and Calcified Areas in Diaphyses and in Arthritis Deformans, Surg., Gynec. & Obst. **68**:129-146, 1939.

11. Herzmark, M. D.: Bone Changes in Caisson Disease, Bull. Hosp. Joint Dis. **3**:128-133, 1943.

12. James, C. C. M.: Late Lesions in Caisson Disease, Lancet **2**:6-8, 1945.

13. Taylor, H. K.: Aseptic Necrosis in Adults, Caisson Workers and Others, Radiology **42**:550-569, 1944.

14. Bell, A. L. L.; Edson, G. N., and Hornick, N.: Characteristic Bone and Joint Changes in Compressed-Air Workers, Radiology **38**:698-707, 1942.

15. Rendich, R. A., and Harrington, L. A.: Roentgen Findings in Caisson Disease of Bone with Case Reports, Radiology **35**:439-448, 1940.

16. Walker, W. A.: Aseptic Necrosis Occurring in Caisson Disease: Case, J. Bone & Joint Surg. **22**:1080-1084, 1940.

cited in which there were roentgenologic changes identical with those found in caisson disease without any history of the patient having worked in compressed air or having been subjected to sudden and violent changes of atmospheric pressure. Taylor [13] mentioned that he has collected a series of 41 cases in which there were lesions of the shafts and joints identical in appearance in many instances with those described in caisson disease but in which the history failed to disclose any known etiologic factor to account for the roentgen findings, and he has suggested the possibility that the bone infarct sometimes seen in men working under compressed air may have antedated the occupation and may bear no relation to it.

Fig. 2.—Gross changes in animal subjected for thirty-three hours to compression at 55 pounds per square inch and killed one month later. The microscopic changes were similar to those in figure 5.

The experimental work in this paper has been directed toward the production in animals of symptoms and early lesions that simulate the later lesions of the bone seen in roentgenograms made on certain caisson workers.

APPARATUS AND METHODS USED

A pressure chamber was used (fig. 1) which was 38 inches (91 cm.) in length and 32 inches (81 cm.) in circumference and which was attached to a compressed air tank and had a total capacity of 200 liters of air compressed to 1,800 pounds (816 Kg.) per square inch (42 sq. cm.).

A regulation reduction valve was attached to the tank of compressed air, and there was another gage attached to the pressure chamber. The gages measured pounds per square inch. The pressure chamber was fitted with two windows for observation.

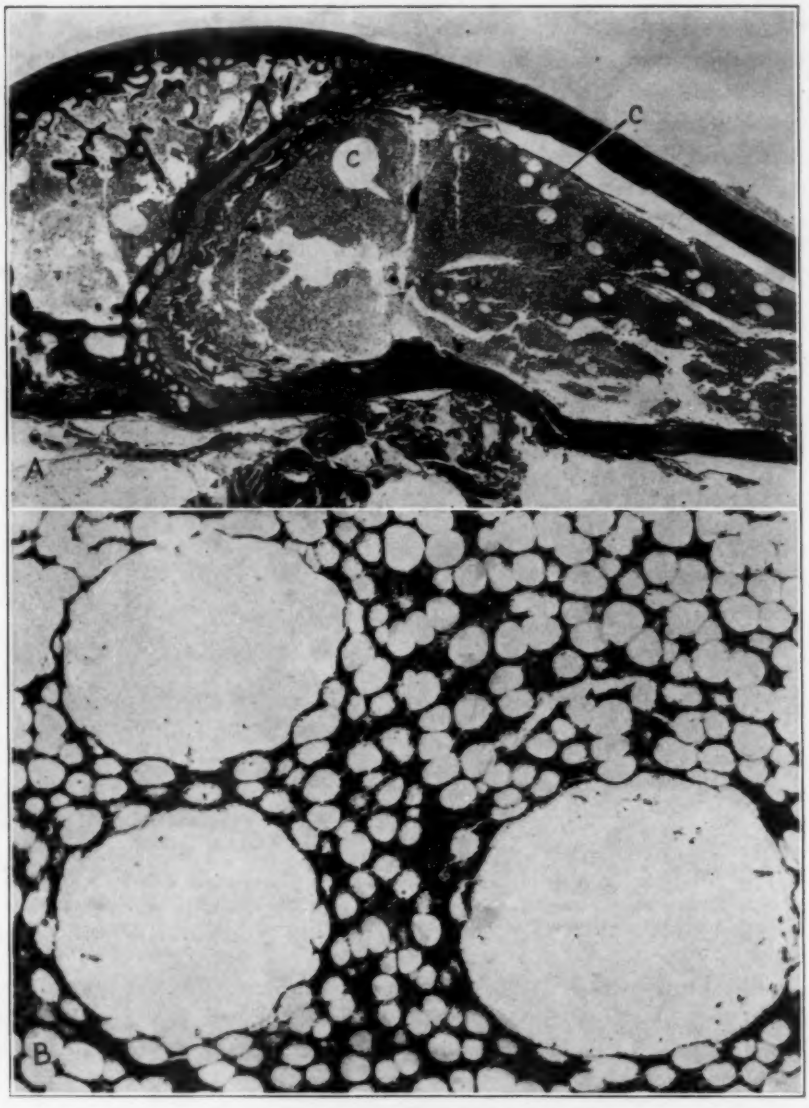

Fig. 3.—*A*, cross section of the femur in an animal that was compressed for six and a half hours at 60 pounds of pressure per square inch and died immediately after decompression. There are many cavities varying in size from 0.5 to 1.5 mm. in the soft tissue of the medullary canal. Some are lined with a thin layer of pink-staining material resembling edema or tissue fluid. Others have no lining and appear to have replaced or pushed aside the soft medullary tissue. This is further evidenced by the adjacent tissue surrounding the periphery of the cavity; × 4.5. *B*, microscopic picture of the area in *A*, showing cavities which represent replacement of tissue by air bubbles following decompression; × 120.

After several months of trial the optimum amount of compression was established at 55 pounds (25 Kg.) of pressure per square inch maintained for five and a half hours. In order to determine the optimum amount of compression animals were placed in the compression chamber for periods of increasing length with increasing pressure. For example, the first animal was compressed for thirty

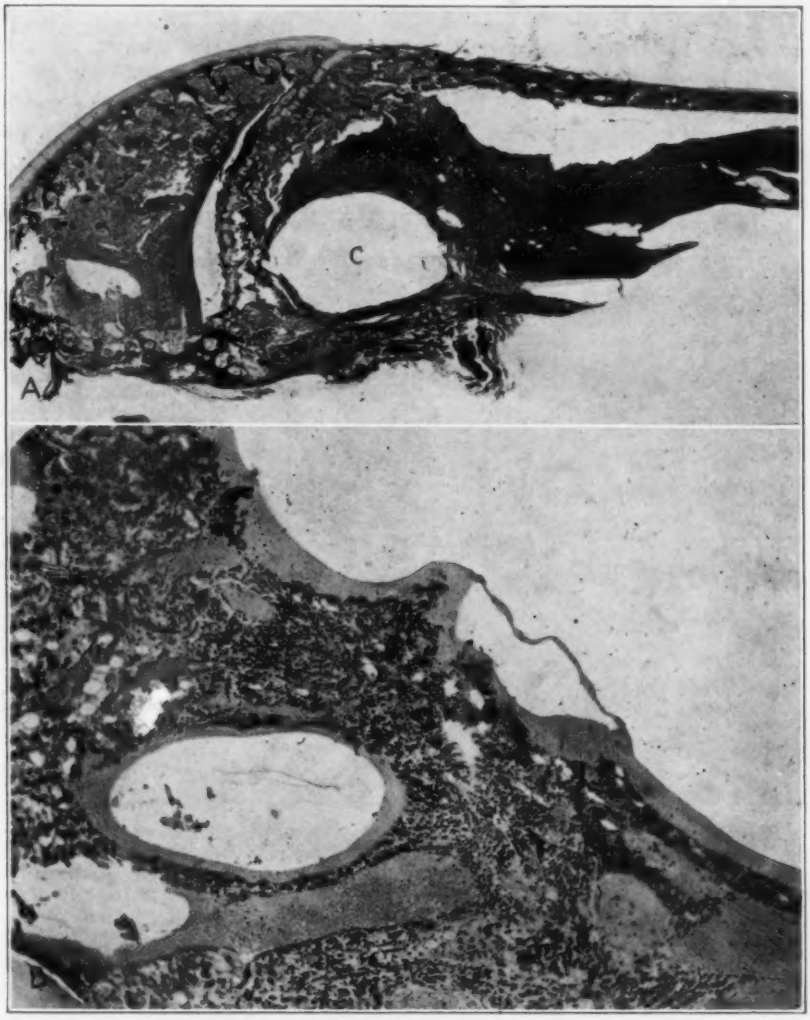

Fig. 4.—*A*, sagittal section of the femur of an animal compressed for ten hours at 55 pounds per square inch over a period of a week. Autopsy was performed one month later. A large cavity is shown in the medullary portion of the femur measuring 8 by 5 cm. There is a pink-staining lining to the cavity, which represents tissue fluid or edema; × 4.5. *B*, microscopic picture of the large cavity in *A*; × 120.

minutes at 40 pounds (18 Kg.) of air per square inch, which is somewhat less than three atmospheres of pressure. The period was finally increased to five

and a half hours of compression at 55 pounds (25 Kg.) of air per square inch, or slightly greater than three and a half atmospheres of pressure, and this was found to be the critical point, at least as far as rabbits were concerned. This was compatible with life in most instances and insured a considerable amount of formation of bubbles in the blood vessels and tissues after decompression.

Adult rabbits were placed in the pressure chamber and the pressure was slowly raised to 55 pounds (25 Kg.) per square inch and then maintained for five and a half hours. After this the pressure was rapidly released and "the bends" allowed to develop in the animals. This was repeated until the animal had undergone compression for thirty-three hours, and then it was killed. Compression higher than 55 pounds per square inch and for longer than five and a half hours usually resulted in death of the animals in from several minutes to one hour after their removal from the chamber.

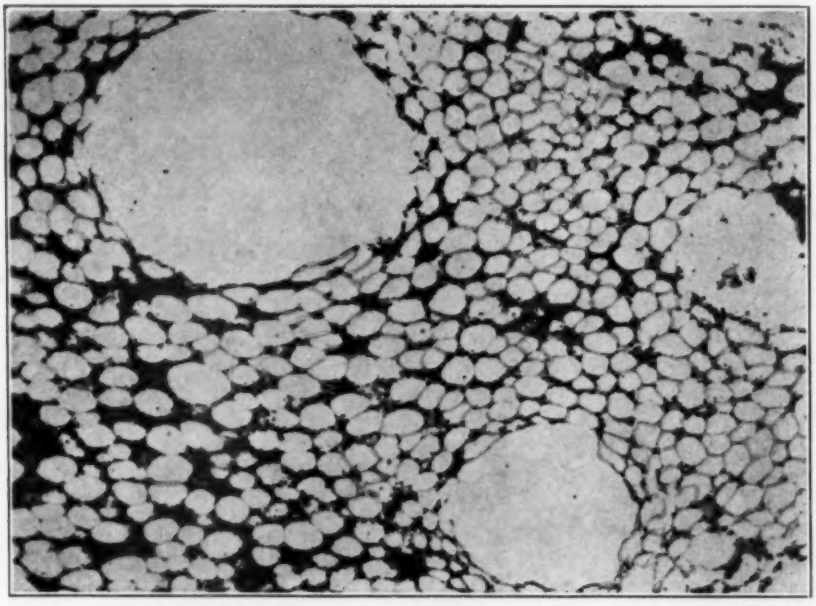

Fig. 5.—Large areas in the medullary cavity, similar to those described in the other figures, of an animal which underwent compression for thirty-three hours at 55 pounds and was killed two months later. The reaction of the tissue to these cavities was minimal after two months, and no evidence of revascularization was seen; × 120.

Twelve animals were used, the amount of compression ranging from 30 pounds (13.6 Kg.) per square inch for two hours to 55 pounds (25 Kg.) per square inch for thirty-three hours. One animal for which the compression was raised to 60 pounds (27 Kg.) per square inch for six and a half hours died on removal from the chamber. Roentgenograms of the animals were taken before the original compression and after the completion of the experiment.

RESULTS

A. Gross Changes.—Autopsy on animals that died directly after compression of acute caisson disease revealed numerous air bubbles in

the venous circulation, fat, subcutaneous tissues, mesentery, fascial planes and mediastinum. The lungs were congested and the heart dilated.

Animals on which autopsy was performed one month after completion of a series of compressions had large cavities in the medullary canal of some of the long bones, with evidence of old hemorrhage (fig. 2). The cortex showed no changes.

B. Microscopic Changes.—In animals that died after decompression there were numerous cavities in the soft tissue of the medullary canal of the long bones. These cavities were lined with a pink-staining material, thought to be edema or tissue fluid. The adjacent peripheral tissue was compressed. Scattered throughout the tissues were areas of frank hemorrhage and strands of fibrin. There were edema and congestion of these tissues. There were no cortical changes. These hollow spaces were interpreted as being the result of release of air bubbles in the tissues following decompression (fig. 3).

In animals on which autopsy was performed one month after completion of the series there was essentially the same picture, with evidence of congestion of the tissues (fig. 4). No definite cortical lesions were found.

An animal subjected to autopsy two months after completion of compression at 55 pounds per square inch for thirty-three hours had the characteristic cavities in the medullary canal (fig. 5).

SUMMARY

1. The factor or factors producing the fundamental change in the tissues leading to the clinical development of aseptic necrosis of bone are frequently unknown. However, it is known that its recognition at present is by means of the roentgenogram—a crude medium for its early detection.

2. In the experiments discussed, cavities or hollow spaces were consistently found in the bone marrow grossly and microscopically. These are interpreted as representing areas which were formed by large nitrogen bubbles collecting in the soft marrow of the long bones. These areas appeared after repeated decompression and produced a permanent defect in the tissues of the medullary canal long after the nitrogen bubbles were absorbed. They are interpreted as representing early changes found in the bone in caisson disease.

3. The lesions of the shafts and joints as demonstrated by roentgen examination require a considerable time to develop in human beings, and it is felt that the changes in the bones of rabbits represent an early stage in the production of aseptic necrosis of bone.

ABSTRACT OF DISCUSSION

DR. DALLAS B. PHEMISTER, Chicago: As Dr. Colonna has pointed out, there is much to be learned about the etiology of aseptic necrosis of bone.

The solitary lesions are most frequently produced by injury, especially by fractures bordering on joints, dislocations and slipped epiphyses, in all of which the blood supply is severed.

The causes of multiple aseptic infarcts of bone are poorly understood, with the exception of those occurring in caisson disease. In the latter condition large infarcts may be formed in either the shaft or the articulating ends of the bones of the large extremities, especially the proximal end of the femur and humerus. Rapid release of nitrogen gas in the tissues from too rapid emergence from the compression chamber may create nitrogen emboli which block bone arteries. Another possibility is that the bone marrow fat which absorbs a proportionately large amount of nitrogen may release it in the tissues, with resultant compression and asphyxia of the intramedullary and cancellous bony structures. Dr. Colonna's findings of large to small cavities within the bone marrow may be an indication of pooling of the nitrogen gas bubbles liberated from the fatty marrow.

From a study in our laboratory of old bone infarcts in caisson disease at autopsy, there has been no evidence of such pocket formation. The areas have consisted of dead cancellous bone and bone marrow, and evidences of invasion and substitution by new bone have been seen about the periphery. After many years, replacement is arrested and the invading fibrous zone and interior of the infarct may calcify. Areas bordering on the hip or shoulder joint may collapse from weight bearing.

Further experimental studies are indicated in order to determine just how the type of massive infarct encountered clinically is produced.

DR. GEORGE O. EATON, Baltimore: It is indeed an honor to be invited to discuss Dr. Colonna's excellent paper. Since Dr. Phemister has studied and written so much on the subject of aseptic necrosis my remarks will be brief. This subject has attained prominence in the past few years because the treatment of fractures of the neck of the femur has improved so much that now we are concentrating on eliminating the occurrence of the all too frequent complications, of which aseptic necrosis is the least predictable and therefore perhaps the most disappointing.

Necrosis is defined as death of a circumscribed portion of tissue. It is seen in various organs such as the pancreas and liver, in fatty tissues and in bones; it may occur in any tissue. In all cases the common factor apparently is lack of sufficient circulation to sustain the life of the tissues. The most common location of this lesion in orthopedic practice is in the head of the femur. One occasionally sees it in tarsal and carpal bones. In the head of the femur it most commonly follows fracture of the neck of the femur or dislocation of the hip. The incidence is high enough so that the prognosis must always be guarded.

The occurrence of aseptic necrosis as a complication of caisson disease has already been brought to our attention in the literature. Dr. Colonna's work is a step forward in the study of aseptic necrosis. More information is needed to explain the sudden and rapid course of the changes in some cases and the comparatively late and slow changes seen in other apparently similar cases. Procedures or technics which will allow us to avoid or minimize the permanent deformities of

the head of the femur may be developed when we have a better understanding of all the factors involved in aseptic necrosis.

Dr. E. D. Jones, Philadelphia: This experimental work was started with the idea of attempting to produce early bone lesions resembling those seen in caisson disease.

The lesions described by Dr. Phemister were late results. It is not known whether the early lesions which we have described would in time exhibit the same reaction.

In regard to Dr. Phemister's suggestion that this study should be continued, we are doing this, and the next stage of the experiment will consist in compressing animals and observing them over a longer period, perhaps months or even a year.

We wish to thank Dr. Phemister and Dr. Eaton for their remarks.

TREATMENT OF CARCINOMA OF THE FUNDUS UTERI

LEON S. McGOOGAN, M.D.
AND
HOWARD B. HUNT, M.D.
OMAHA

THE DEPARTMENT of obstetrics and gynecology of the University of Nebraska Hospital was organized in 1931. Since that date, 72 patients with carcinoma of the fundus uteri have been admitted to the service for study and therapy.

Data on Seventy-Two Patients with Carcinoma of the Fundus Uteri

	Years	Patients	Survivors, Five Years	Patients	Survivors, One to Five Years
No therapy					
Terminal	.....	3	0	..	..
Adequate	.....	..	..	1*	..
Roentgen irradiation alone	1937 to 1941	1	0	..	..
Palliative	1942 to 1946	..	..	5	0
Radium	1931 to 1937	6	2	..	..
	1942 to 1946	..	..	1	0
Surgical treatment alone	1931 to 1941	5	2	..	..
	1942 to 1946	..	..	1	1
Adequate surgical treatment plus radium or roentgen therapy	1933 to 1941	4	4	..	..
Inadequate surgical treatment plus radium or roentgen therapy	1931 to 1941	6	1	..	..
	1942 to 1946	..	..	3	1†
Roentgen irradiation and radium	1937 to 1941	12	5	..	..
	1942 to 1946	..	..	14	7‡
Roentgen irradiation, radium and surgical treatment	1937 to 1941	3	3	..	..
	1942 to 1946	..	..	6	5
Radium and surgical treatment	1937 to 1941	1	1	..	..
Totals		41	18 (43.9%)	31	14

* Treated elsewhere.
† Operated on elsewhere.
‡ Three to return for hysterectomy.

Inasmuch as the university hospital is available only to those who are indigent, many of the patients were in the advanced stage of the disease process. Many were from areas which are some distance from the hospital. Follow-up therapy has been difficult in many instances because of the distance of travel involved and the expense of transportation.

Read at the fourth annual meeting of the Central Surgical Association, Chicago, Feb. 22, 1947, by invitation.

From the Department of Obstetrics and Gynecology and the Department of Radiology, University of Nebraska College of Medicine.

The method of therapy for each particular case has been varied or modified in relation to the general condition of the patient, the extent of the disease process and the type of radiotherapy available.

The program of treatment of cancer of the body of the uterus with radium and high voltage roentgen irradiation in the Department of Radiology at the University of Nebraska Hospital is characterized by individualization in each case, fractionation of the radium and correlation of high voltage roentgen irradiation and radium therapy. Treatment has been individualized according to the extent of the disease, the size and location of the primary tumor and the length of the uterine canal as well as to the size and general health of the patient. Treatment by roentgen irradiation alone has been confined to patients with metastases or regional extensions which precluded possible eradication or control of disease. Radium therapy alone was not voluntarily selected for any cases. Deep roentgen irradiation was not used in addition to radium prior to 1937, since no facilities were then available. Since that time, 2 patients received no deep roentgen irradiation merely because radiotherapy was prematurely terminated by the presence of a pulmonary embolus in 1 instance and at the patient's request in the other.

The program of high voltage roentgen irradiation has been varied according to the size, obesity and general condition of the patients. The roentgen rays have been energized by 200 kilovolts constant potential screened through a Thoraeus A combination filter and delivered from a 70 cm. distance. The daily dosage usually consisted of 180 r (measured in air) delivered to each of two portals. A combination of two to six portals has been utilized, so arranged and treated as to deliver a total dosage of about 2,500 to 3,000 r into the midpelvis. This dosage can be delivered into the flat pelvis of a slender patient through one large anterior and one large posterior portal. Three or more portals for delivery of this midpelvic dosage may be required for larger, rotund patients in order not to exceed the amount tolerated by the skin. The dosage delivered over any one cutaneous area has rarely exceeded 2,000 r (measured in air). The midline of the abdomen is protected by a lead strip 2 cm. in width. This serves the dual purpose of shielding the skin along the line of future incision and of slightly reducing the combined high dosage in the immediate vicinity of the area of the intrauterine application of radium. The skin of obese patients tolerates radiation poorly, especially along the groins and the creases in the skin. Elderly and debilitated patients show more nausea, anorexia and depression and likewise recover more slowly from radiation sickness and enteritis than do more robust patients. The febrile patient must be treated cautiously with roentgen irradiation. The intrauterine application of radium is hazardous until after the temperature has been reduced to a normal range. The incidence of pelvic infection during and after

radium therapy has been reduced by preliminary high voltage roentgen therapy, fractionation of radium therapy and avoidance of prolonged intrauterine applications.

Radium has usually been delivered in a series of two to four intrauterine applications spaced about ten to fourteen days apart. Total dosage has varied from 4,000 to 8,600 mg. hours. The usual intrauterine technic has consisted of three more or less equal intrauterine applications of 65 mg. for thirty to thirty-six hours spaced about twelve days apart. Radium has been filtered through 1 mm. of platinum and distributed along the entire length of the uterine canal in a rubber sac. This delivers about 2,500 gamma roentgens to a zone 2 cm. distant from the average uterine canal. High voltage roentgen irradiation has been applied serially during the intervals between the intrauterine applications of radium.

It might be well to explain the motives behind this fractionation of radium and its combination with high voltage roentgen therapy. A massive carcinoma within a large uterus shrinks progressively during constant pounding with the roentgen rays and even more rapidly after each concentrated blow with radium. This progressive shrinkage permits each successive application of radium to approach more and more closely to the basal periphery of the neoplasm. Subintensive fractionated dosage has the further advantage of permitting relatively greater recovery in the intervals of the normal tissues in comparison with the more vulnerable cancerous tissue than is possible with the more severe injury caused by concentrated massive dosage. The concurrent application of high voltage roentgen irradiation and radium therapy permits delivery of a larger effective combined dosage to the zone 2 cm. and more beyond the uterine canal, a region in which radium alone is ineffective. Furthermore, the utilization of fractionated radium in addition to high voltage roentgen therapy does shrink the carcinoma more rapidly and completely than would the latter alone. It is our present practice to make the final application of about half the total dosage of radium ten to fourteen days after completion of the combined high voltage roentgen therapy and preliminary application of radium, since we believe that significant further shrinkage of the tumor will have occurred during the extended interval. We are slow to accept fully the currently popular radiologic tenet that all high voltage roentgen therapy should have been completed and recovery made before any intrauterine application of radium is made. The placement of radium along the entire uterine canal offers the advantages of more even irradiation of the entire uterus and of a greater depth of dosage to the periphery. Furthermore, the danger of necrosis in the uterine wall or of injury to adjacent tissues is lessened by the use of the long applicator.

The surgeon can contribute much to improve the effectiveness of a radiotherapeutic program in carcinoma of the body of the uterus. A

diagnostic curettage done prior to a supracervical hysterectomy for fibromyoma of the uterus may disclose an unsuspected carcinoma of the endometrium. In case carcinoma is present, radiotherapy can be much more effectively administered before hysterectomy has been done than after. Previous hysterectomy precludes any effective application of radium along the uterine canal. Radium limited to the cervical stump and vaginal vault is much less effective. Intrauterine application of radium ensures a larger parametrial dose and eliminates or devitalizes the carcinoma, thereby reducing the hazard of dissemination by the trauma incident to hysterectomy. Any application of radium made at the time of curettement or shortly after should represent not more than one third of the total proposed dosage. The other two thirds of the full dosage will be much more effective when administered after considerable preliminary shrinkage has been brought about by intensive high voltage roentgen irradiation and subintensive radium therapy. Both of these are essential for proper preliminary shrinkage of cancer and for delivery of adequate radiation to the parametrial tissues. The complete extent of disintegration and disappearance of cancer cells does not become fully apparent histologically until eight to twelve weeks after final radiotherapy. Pelvic congestion will have subsided and endometritis will have usually cleared by then. Hysterectomy can be advantageously delayed until after this interval of eight to twelve weeks.

With the addition of high voltage roentgen therapy in 1937, it was thought advisable, after consultation with Dr. H. B. Hunt, the radiologist, to treat as many patients as possible with the following method: After proper diagnosis had been established by a diagnostic curettage, the carcinoma was to be subjected to high voltage roentgen therapy and intracavity radiation, followed in about eight to twelve weeks by surgical treatment, due regard being taken for the general condition of the patient and the extent and operability of the lesion.

Since 1937, 35 patients have been admitted and treated with high voltage roentgen irradiation and intracavity radiation. These patients form the basis for this report. The doses of roentgen rays or radium for each case have been carefully gaged according to the extent of the lesion, the reaction of the patient as demonstrated by gastrointestinal disturbances, i. e., nausea, vomiting, diarrhea and rectal tenesmus, and the reaction of the skin.

Two to three months after the conclusion of the radiation therapy the patient was seen again, and the possibility of surgical removal of the carcinoma was considered. During this period of delay, the results of the radiation become effective. There is a shrinkage or decrease in the size of the lesion, and any infection present is controlled; surgical treatment is thus made easier and the postoperative morbidity and mortality lower. In addition the patient's general physical condition can be improved and

readied for operation. Anemia can be controlled by the administration of iron or by transfusion, obesity lessened by proper diet, diabetes controlled by diet and insulin and cardiac, hypertensive or renal conditions brought under proper management. All this tends to lessen the risk in surgical procedures.

From 1937 to 1941, 15 patients were treated with preliminary high voltage roentgen irradiation and radium. Only 3 were selected for surgical treatment. Of the remaining 12 patients, 7 died from intercurrent disease or from carcinoma within two years, and 5 lived for five years or more. This gives a total of 8 patients who survived, or 53.3 per cent.

From 1942 to 1946 inclusive, 20 patients were treated by the method just described, and 6 were selected for surgical treatment and have been operated on; 3 more are to have surgical treatment. Seven patients not operated on have succumbed to intercurrent disease or carcinoma, and 1 patient operated on died one year later. This is a total of 12 patients surviving for one to five years, or 60 per cent.

In 9 of the 35 cases, therefore, the carcinoma was adjudged operable from both the clinical and the technical standpoint and operation has been carried out. The operation consists of panhysterectomy, that is, removal of the uterus, cervix, ovaries and tubes. Numerous authors have reported difficult operation, friability of tissues, persistent oozing and delay in healing of the wound after radiation. The last can be prevented by protecting the area of the proposed incision by a narrow lead strip during all exposure to high voltage roentgen irradiation.

Technical difficulty was encountered only once and necessitated the leaving of a small cervical stump. The operative procedure of panhysterectomy was easily accomplished in the remaining 8 cases. The wounds of the skin and vagina healed easily and by primary union. No primary surgical mortality was encountered. In 1 patient treated with intrauterine application of radium hysterectomy was also performed.

Nine of the ten uteri and appendages that were removed were subjected to careful pathologic examination, and in only 1 instance was there any evidence of residual carcinoma, an incidence of 12.5 per cent.

One patient has died since her operation, the exact cause being unknown. Permission for autopsy could not be obtained. One patient has survived for nine years, 2 for seven years, 1 for six years, 1 for four years, 3 for three years and 1 for two years.

Within the past four months, three excellent reviews of the therapy of carcinoma of the fundus uteri have been presented by Scheefey,[1] Miller[2] and Taylor and Becker.[3] Scheefey and others in 1935 began to

1. Scheefey, L. C.: Am. J. Obst. & Gynec. **52**:529-539, 1946.
2. Miller, N. F.: Am. J. Obst. & Gynec. **52**:894-903, 1946.
3. Taylor, H. C., Jr., and Becker, W. F.: Surg., Gynec. & Obst. **84**:129-139, 1947.

treat patients with 4,500 mg. hours of intracavity radiation followed by surgical intervention whenever possible. They reported a five year survival rate of 90 per cent among a group of 10 patients, with 20 additional patients among a group of 21 surviving for one to four years. In seventeen of the uteri removed (54.8 per cent) residual carcinoma was found.

Miller and his co-workers used high voltage roentgen therapy followed by surgical intervention. Ninety-six patients were treated between 1931 and 1945, with a five year survival of 77 per cent and 1 postoperative death. Eighty-one, or 84.4 per cent, of the uteri removed revealed residual carcinoma.

Taylor and Becker reported on 31 patients treated with hysterectomy and radium for whom the incidence of "five year cures" was 64.5 per cent, 13 patients treated with hysterectomy and radiation therapy for whom the incidence was 38.4 per cent and 74 patients treated with combined roentgen and radium therapy and hysterectomy for whom the incidence was 44.6 per cent. The high voltage roentgen therapy apparently was given postoperatively instead of preoperatively.

Although the presented series of 9 cases is too small from which to draw conclusions, it is offered with the hope that the results will stimulate other centers of therapy to investigate the possibilities of combined roentgen therapy and intracavity radiation followed by surgical intervention as a means of obtaining a higher salvage of patients affected with carcinoma of the fundus uteri.

UNUSUAL PROLIFERATIVE REACTIONS TO SUTURE MATERIAL

JOSEPH A. WITTER, M.D.
DETROIT

APPROXIMATELY one year ago the interest of my colleagues and me in the reaction to suture material was renewed and stimulated by a somewhat trying postoperative incident. The patient was a 47 year old white woman who was admitted to the hospital with a diagnosis of uterine fibroids. A total hysterectomy and a bilateral salpingo-oophorectomy were performed according to our usual technic. Cotton suture material was used throughout. The immediate postoperative course was uneventful except that on the eighth day two small blebs appeared in the incision. These were opened, and yellowish, clear serum escaped. During the following weeks, several other blebs developed, and each resulted in a small draining sinus. Concomitant with this, the tissue adjacent to the incision became indurated, without other evidence of inflammation. Soon cotton knots began to be extruded, but the condition did not subside. As months passed, the patient began to have difficulty flexing the trunk because of stiffness of the abdominal wall; also there was progressive frequency of urination, without dysuria. All types of therapy were tried, but nothing seemed to stop the progress of the lesion. Eventually obstruction of the bowel developed, and the patient was again submitted to surgical treatment. At operation the abdominal wall and underlying viscera appeared to be infiltrated by a dense neoplastic process, which on close inspection seemed to be intimately related to the previously placed cotton. It was necessary to resect 6 inches (15 cm.) of small bowel, the dome of the bladder and a strip of abdominal wall 2 inches (5 cm.) wide. Surgical gut and wire sutures were used. The pathologist reported diffuse fibrosis and reaction to a foreign body. The patient made a complete recovery.

Such a severe reaction, apparently due to suture material, cannot be regarded lightly. Accordingly, a survey was made at our hospital of all patients whose wounds were sutured with cotton during 1946, and the known reactions were tabulated. Reactions other than the one just described consisted of formations in the sinus tract and did

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not jeopardize the life of the patient in any way. Some of these sinuses healed after extrusion of knots, while others required surgical excision.

We were fortunate in being able to obtain the figures on a similar series from another hospital in Detroit.[1] These paralleled ours in all respects, and included among the reactions was a condition which resembled a connective tissue neoplasm.

Table 1 gives the totals for these two series. In the group at the Highland Park Hospital there were 341 cases in which cotton was employed as the suture material in major operations. Eleven reactions took place, of which one was serious and the remainder were in the form of a draining sinus tract. The incidence of reactions was 3.2 per cent.

TABLE 1.—*Total Reactions*

Hospital	Cases	Reactions	Percentage of Reactions
Highland Park General	341	11	3.2
Harper	1,000	30	3.0

TABLE 2.—*Analysis of Highland Park Hospital Series*

Operations After Which Reactions Occurred	Number of Reactions
Cholecystectomy	2
Herniorrhaphy	1
Salpingectomy	1
Appendectomy	2
Hysterectomy	4
Gastrectomy	1
Total	11

The series at the Harper Hospital consists of 1,000 cases, with reactions occurring in 3 per cent, one of which was serious.

Table 2 shows the types of operations after which reactions occurred in the series at the Highland Park Hospital. It is of considerable interest that these operations all were classed as clean except one appendectomy prior to which perforation had occurred. This factor gave some support to our contention that infection is not essential for reaction to suture material.

The accompanying photomicrographs have been taken from representative pathologic sections of tissue removed at secondary operations in the Highland Park Hospital series.

Figure 1 shows a strand of homogeneous material, which we have proved to be cotton, completely surrounded by macrophages, some of

1. Ashley, L. B.: Personal communication to the author.

which are fusing to form syncytial foreign body giant cells. It should be noted that the reaction is not acute and that there is no evidence of infection. If the history of this section were not known, it would be easy to diagnose the condition as a polymorphous cell sarcoma.

By way of contrast, figure 2 demonstrates a section in which there is active inflammation and formation of granulation tissue as manifested by the presence of polymorphonuclear cells, lymphocytes, young capil-

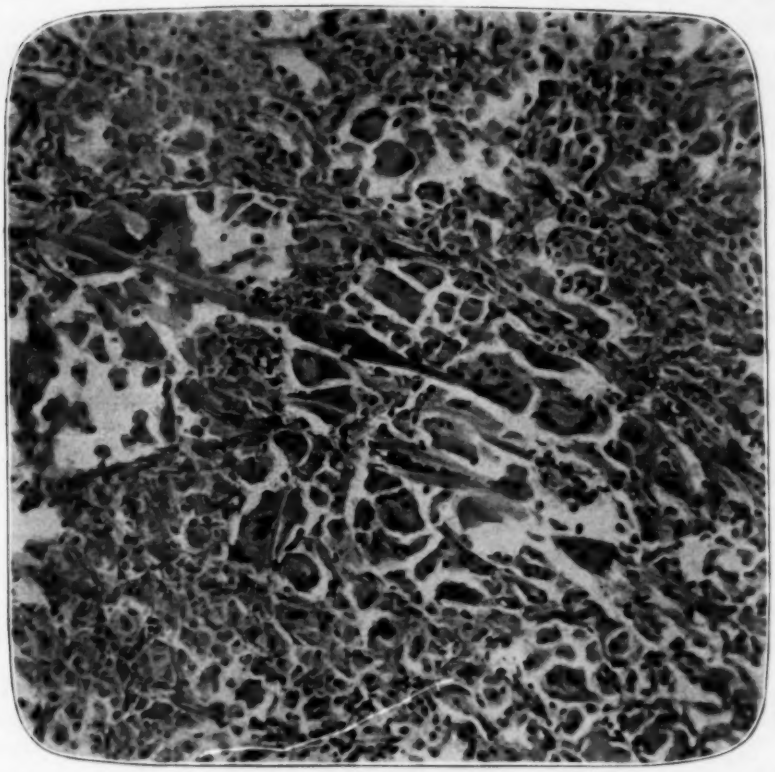

Fig. 1.—Photomicrograph showing cotton fibers surrounded by foreign body giant cells.

laries and fibroblasts. Cross sections of cotton fibers are seen in the center of the section.

Figure 3 reveals mostly old connective tissue which extends for 2 cm. on either side of the suture although the field of this photomicrograph is not great enough to show it all. Fairly close to the suture there is active fibroplasia which gives rise to concentric enlargement of the zone of reaction.

Figure 4 contains bundles of striated muscle in cross section which are undergoing invasion and replacement by fibrous connective tissue.

The cotton suture material which apparently gave rise to this relatively benign invasion was just outside the field of this photomicrograph.

From figure 5 it will be noted that the body is not entirely unable to cope with the presence of buried cotton. Careful scrutiny will reveal that many of the macrophages have engulfed fragments of cotton. It is a matter of record that at an occasional secondary operation no evidence of previously placed cotton can be found. We believe that in most instances, however, the cotton remains at least partially intact for many years.

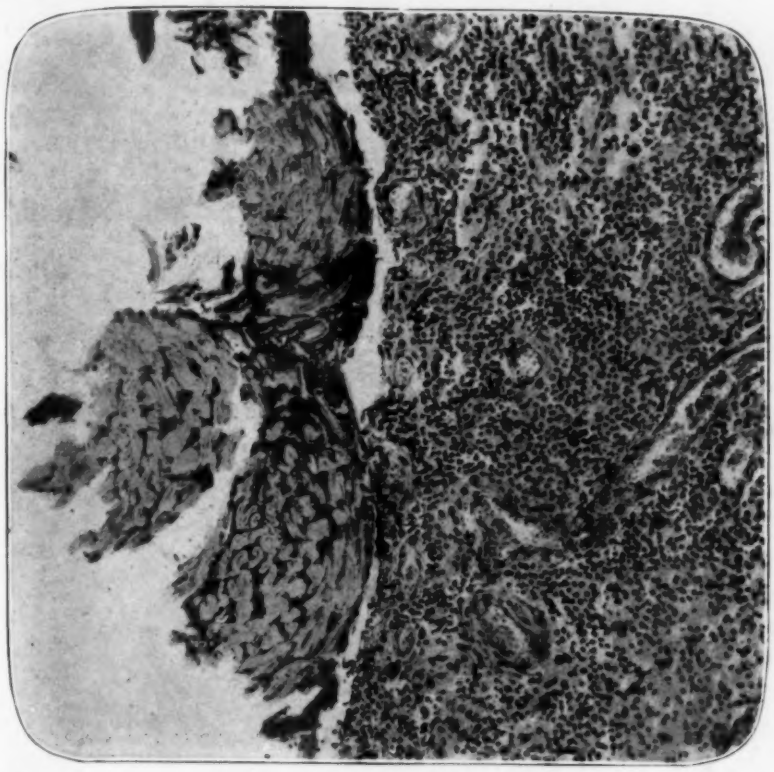

Fig. 2.—Photomicrograph showing cotton fibers surrounded by granulation tissue.

In brief recapitulation of the histologic picture, it may be pointed out that instead of the usual formation of a thin fibrous wall about cotton sutures there is, in 3 per cent of cases, an extreme reaction accompanied with a marked fibroplasia which seems to be the result of continuous stimulation by the cotton or some substance in it. This proliferation has a mildly invasive character, and the adjacent tissues become involved in a dense cicatricial change. It is our impression that

infection is introduced into these reactions secondarily and does not play an etiologic role.

Silk and surgical gut have been used for several decades, and during this time many improvements and refinements have been made. Cotton, on the other hand, is relatively new, and most surgeons have been content to use commercial spool thread. When it is considered that the manufacturer gave no thought to the possible effect that his product might have when buried in human tissues, it will become apparent that cotton should not be condemned hastily. This brings us to the

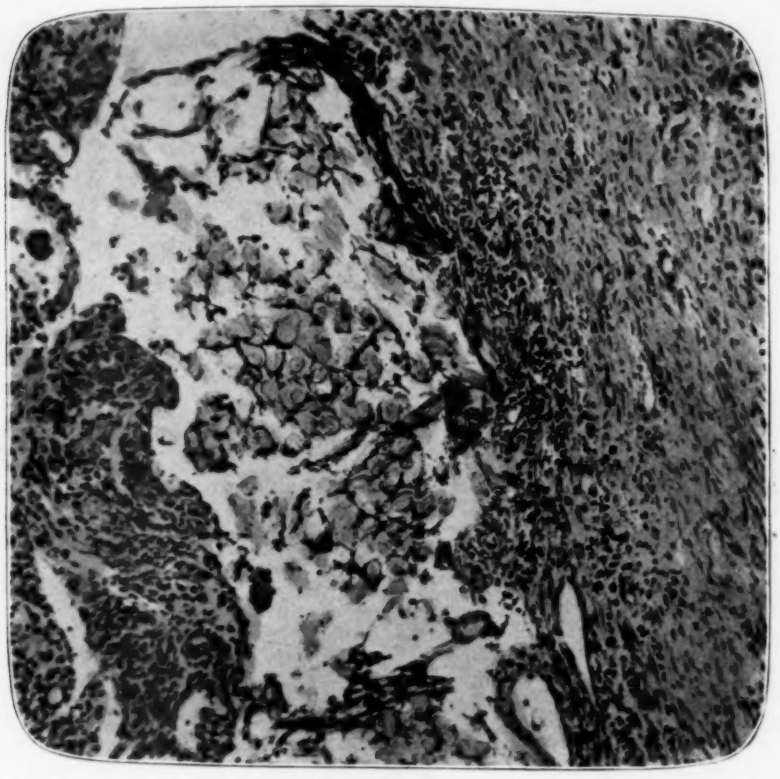

Fig. 3.—Photomicrograph showing cotton fibers surrounded by inner zone of granulation tissue and outer zone of proliferating fibrous connective tissue.

most important issue in the present discussion—the question of what characteristic of spool cotton gives rise to occasional unfavorable reactions. There does not seem to be an allergy in the strict sense of the word, and there is no unusual eosinophilia to be observed in the microscopic sections. Furthermore, cotton has rarely been found guilty as a source of sensitivity. D'Ingianni [2] has stated that he does not feel

2. D'Ingianni, V.: Personal communication to the author.

that the reactions which occur with cotton are due to the presence of the cotton itself, although Searcy [3] and others have shown in experiments on animals that there is a greater fibrous reaction to cotton than to any of the other ordinary suture materials. It is known also that diffuse pulmonary fibrosis will sometimes develop in workers in cotton mills because of continued inhalation of cotton dust. However, it would seem reasonable to assume that if cotton fibers themselves were acting as a continuous irritant the incidence of reaction would be much

Fig. 4.—Photomicrograph of striated muscle in cross section, showing extensive replacement by fibrous connective tissue.

greater than 3 per cent. Derbyshire [4] has recently reported an incidence of 4.5 per cent.

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3. Searcy, H. B.; Carmichael, E. B., and Wheelock, M. C.: Implant Material: Production of Fibrous Tissue Ground Fibers of Cotton and Other Foreign Material Implanted Subcutaneously in Rats, South. M. J. **37**:149-150 (March) 1944.

4. Derbyshire, R. C.: Complications Following the Use of Cotton as Suture Material, Surg., Gynec. & Obst. **84**:31-35 (Jan.) 1947.

Loofbourow [5] has described proliferation-promoting intercellular hormones and has stated certain evidence for their production by living cells in response to injury. If such hormones could be proved to exist, the possibility of cotton acting as a catalyst in their production might be considered.

The most probable explanation, however, would seem to lie in the substances used in the preparation and manufacture of cotton. This would introduce various detergents, alkalis, extractives, mordants and

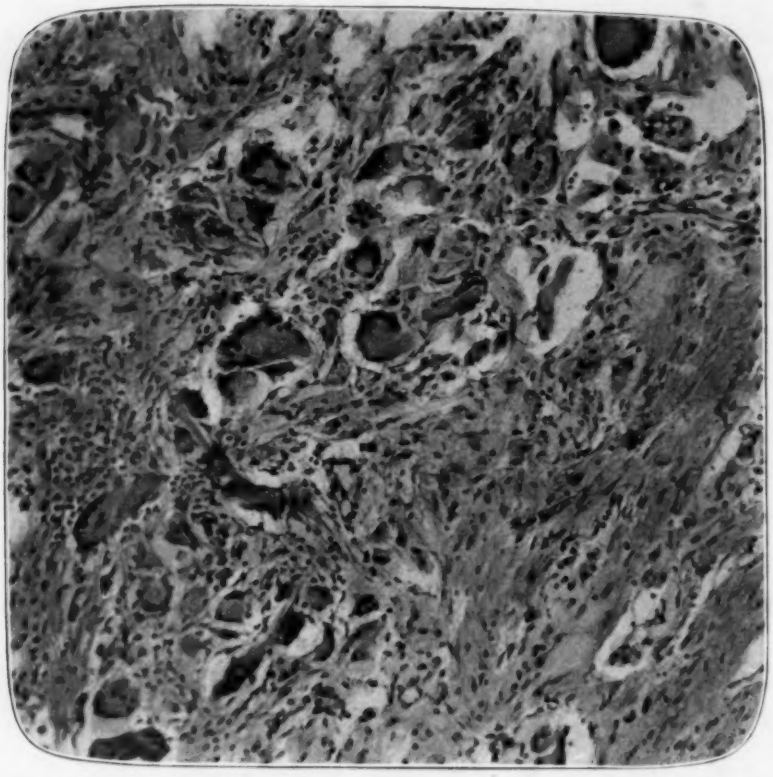

Fig. 5.—Photomicrograph showing fragments of cotton fibers engulfed by foreign body giant cells.

dyes and would require a careful study of the process involved in producing the finished thread from the cotton plant, with biologic experiments at each stage. This will be taken up in future studies and might well become a problem in original research for a resident surgeon working

5. Loofbourow, J. R.: Intercellular Hormone: Further Observations Regarding Mechanism of Production and Release of Proliferation-Promoting Factors by Injured Cells, Biochem. J. **36**:631-638 (Sept.) 1942.

toward his master's degree in surgery. It is to be hoped that the makers of cotton thread will become sufficiently interested in the project to divulge any secrets of processing and to aid the study whenever possible.

At any rate, it will have to be allowed that the preparation of cotton requires the use of many reagents and that each of these reagents is a possible source of irritation. It is also necessary to assume that in some batches of cotton there are traces of these irritants remaining in the product which appears on the market and that it is because of these occasional batches that reactions arise.

This seems an opportune moment to make a plea for a cotton suture especially prepared for surgical use which would be chemically free of all irritant substances and would be composed of selected long fiber plants so as to be more uniform in tensile strength. This is not an unreasonable demand, and the field would seem to hold an ample return of business for any firm which would undertake it. Only when such a cotton suture has been developed can it be fairly compared with other sutures, and only then can it be condemned if it falls short of the standards.

SUMMARY

Two series of cases, totaling 1,341 operations in which cotton was used as the suture material, are presented.

Forty-one patients (3 per cent) reacted unfavorably to cotton. Two (0.1 per cent) of these reactions were sufficiently serious to threaten the life of the patient.

Infection was present in only 1 case in this series and is thought not to play a part in the cause of the reactions.

In certain cases the stimulus to fibroplasia is not self limited, and the resulting fibrosis grossly resembles a neoplastic process.

It is felt that this unfavorable feature of cotton is more apt to be due to the chemicals used in processing than to the cotton itself. Further studies are proposed.

A plea is made for cotton thread especially prepared for surgical use.

PHLEBOTHROMBOSIS ASSOCIATED WITH MUCIN-PRODUCING CARCINOMAS OF THE TAIL AND BODY OF THE PANCREAS

A Clinicopathologic Study of Two Cases with Necropsy

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SANTA BARBARA, CALIF.

THE REPORTS of Whipple,[1] Brunschwig[2] and Waugh and others[3] concerning successful surgical extirpation of the pancreas have served to modify the heretofore hopeless attitude of the medical profession regarding malignant tumors of this organ. Furthermore, these accomplishments have stimulated interest in the early diagnosis of carcinoma of the pancreas, particularly in cases in which jaundice plays an inconspicuous part. Several investigators in the field of pancreatic disease have called attention to pain as one of the earliest and most reliable symptoms in cases of carcinoma of the body and tail. Ransom[4] studied 16 such cases and found epigastric pain to be the predominant symptom. He described the following three types of pain as typical of the disease: (1) a dull aching pain which often is referred to the back, (2) paroxysmal attacks of pain at the umbilical level, also referred to the back and (3) pain in the right upper abdominal quadrant resembling that in biliary colic. In Duff's[5] analysis of 19 cases of carcinoma involving the body and tail of the pancreas he recorded pain in the upper abdominal area as the most frequent complaint.

Read at the fourth annual meeting of the Central Surgical Association, Chicago, Feb. 22, 1947.

From the Division of Surgery and the Division of Pathology of the Santa Barbara Cottage Hospital and the Department of Pathology of the University of Southern California Medical School.

1. Whipple, A. O.: Observations on Radical Surgery for Lesions of the Pancreas, Surg., Gynec. & Obst. **82**:623-631 (June) 1946.

2. Brunschwig, A.: Radical Resection of Advanced Intra-Abdominal Cancer: Summary of Results in One Hundred Patients, Ann. Surg. **122**:923-932 (Dec.) 1945.

3. Waugh, J. M., and others: Total Pancreatectomy: A Symposium Presenting Four Successful Cases and Report on Metabolic Observations, Proc. Staff Meet., Mayo Clin. **21**:25-46 (Jan. 23) 1946.

4. Ransom, H. K.: Carcinoma of Body and Tail of Pancreas, Arch. Surg. **30**:584-606 (April) 1935.

5. Duff, G. L.: Clinical and Pathological Features of Carcinoma of Body and Tail of Pancreas, Bull. Johns Hopkins Hosp. **65**:69-98 (July) 1939.

Sproul[6] was the first to call attention to the frequent association of multiple phlebothromboses with malignant pancreatic tumors in which the site of the primary tumor was distal to the head. Her published observations in 125 cases of carcinoma with associated venous thrombosis resulted in the discovery that in 56.2 per cent of the cases in which the primary tumor was in the body and tail one or more organized venous thrombi were found. In 31.3 per cent of these cases widely disseminated thrombi were demonstrated. Moreover, in the total cases studied there were but 4 instances of multiple phlebothromboses in which the primary site of the tumor was not in the pancreas.

The most recent contribution to the knowledge of this interesting syndrome was furnished by Kenney,[7] who reported 21 cases of carcinoma of the body and tail of the pancreas. The cases were collected from the records of 9,800 consecutive necropsies, and there was associated multiple venous thrombosis in 33.3 per cent.

The purpose of this communication is the addition of 2 cases to the expanding list of cases of malignant pancreatic tumors in which the syndrome under discussion has been observed. Although in the majority of cases studied the thrombotic tendency was first discovered at necropsy, in several instances the symptoms of peripheral venous thrombosis constituted the patient's initial complaint. Inasmuch as the necropsies have shown that the thrombi in such instances are independent of metastatic tumor, it is to be hoped that signs of unaccountable phlebitis or phlebothrombosis may prove an early clue to the existence of a disease which heretofore has been considered hopelessly advanced when first discovered.

REPORT OF CASES

CASE 1.—A 62 year old white woman was admitted to the Santa Barbara Cottage Hospital on Feb. 22, 1946, complaining of pain and swelling of the left leg.

Present Illness.—Three weeks before her admission she complained of moderately severe pain in the calf of the left leg. After two days of rest in bed the entire leg became swollen and tender. Five days later she had a sharp pain in the left side of the chest associated with dyspnea. Several days after this similar pain was observed in the right side of the chest. No cough was associated with the pain, and after a week of rest in bed it had partially subsided. Persistence of the painful swelling in the leg brought the patient to the hospital.

Past History.—The past history was essentially noncontributory except that in recent years the patient had suffered from occasional cramping pain in the left leg.

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6. Sproul, E. E.: Carcinoma and Venous Thrombosis: Frequency of Association of Carcinoma in Body or Tail of Pancreas with Multiple Venous Thrombosis, Am. J. Cancer **34**:566-585 (Dec.) 1938.

7. Kenney, W. E.: Association of Carcinoma in the Body and Tail of the Pancreas with Multiple Venous Thrombi, Surgery **14**:600-609 (Oct.) 1943.

Physical Examination.—Examination revealed a well developed and well nourished white woman in no obvious distress. The temperature was 100.4 F. and the pulse 82 beats per minute. Respirations were 18 per minute, and the blood pressure was 180 systolic and 80 diastolic. There were no abnormal findings relating to the head and neck. Dulness to percussion was noted posteriorly over the lower part of the left lung. Inspiration evoked moist rales in the axillary line. Examination of the heart and the abdomen disclosed nothing remarkable. Both lower extremities were edematous, and the right leg was colder to touch than the left.

Laboratory Examination.—On admission of the patient to the hospital the erythrocyte count was 5,090,000, and the hemoglobin content was 13.5 Gm. per hundred cubic centimeters. The white blood cell count was 23,450, with a normal differential count. The prothrombin time as determined by the Quick method was 101 per cent. Examination of the urine showed it to be normal, and reactions to the Kline and Kolmer serologic tests for syphilis were negative.

Clinical Course.—A diagnosis of phlebothrombosis of the left femoral vein, with probable involvement of the inferior vena cava at the level of its bifurcation, was made when the patient was admitted to the hospital. She was operated on two hours after her admission, a bilateral femoral ligation being performed. A partially organized thrombus 9 cm. in length was removed through the incision in the left femoral vein. The day after the operation the patient complained of numbness in the right lower extremity, but the color in both legs remained good and the edema disappeared. "Dicumarol" (3,3'-methylene-bis-4-hydroxycoumarin) therapy was begun, with the patient receiving an initial dose of 300 mg. of the drug. Two days later the right leg became swollen and slightly cyanotic and the calf was tender on palpation. This condition persisted in the right leg, and on March 6 pitting edema again appeared in the left leg. At this time the patient had shortness of breath. The temperature remained elevated. Examination of the blood on March 15 revealed an erythrocyte count of 3,600,000, a hemoglobin content of 10.5 Gm. per hundred cubic centimeters and 8,000 white blood cells. Subsequently, there was little change in her condition except that the legs became more edematous and respiratory embarrassment was more pronounced. The course was gradually downhill, and the patient died on April 7, 1946, forty-three days after her admission to the hospital.

Observations at Necropsy.—Permission was granted for examination of the thoracic and abdominal organs and the bone marrow. The significant observations were as follows:

External examination of the body disclosed slight icterus of the skin and scleras. The peritoneal surfaces were free, smooth and glistening. Palpation of the pancreas disclosed a firm, diffuse, moderate enlargement of the tail. The liver contained numerous nodules of tumor varying from a few millimeters to 2 cm. in diameter. The weight of the pancreas was moderately increased, being 150 Gm. The tail was indurated and moderately enlarged and of a lighter color than the normal-appearing pancreatic tissue. Section through the organ disclosed that the tail of the pancreas in the area of the previously noted induration measured 5 cm. in length and 4 cm. in width (fig. 1*A*). In this area the pancreatic tissue was completely replaced by a firm gray-white and pink tumor that diffusely infiltrated the adjacent parenchyma of the body. No clear line of demarcation between normal gland and tumor could be made because the two merged imperceptibly. Careful examination of the cut surface showed small areas of gray-yellow necrosis, measuring up to 2 mm. in diameter.

The weight of the liver was slightly reduced, being 1,150 Gm. Externally, the surface was covered with gray-white and pink nodules of tumor varying from

a few millimeters to 4 cm. in diameter. Section disclosed nodules of tumor in all parts of the liver. The tumor in the liver was identical in gross appearance to the tumor observed in the pancreas. Several enlarged firm lymph nodes filled with the same type of tumor surrounded the pancreas, some measuring up to 2 cm. in diameter. The inferior vena cava was filled with a partially organized black-red thrombus which extended 8 cm. above the bifurcation (fig. 1 *B*). The thrombotic process involved both iliac veins and the femoral and ovarian veins. The splenic vein contained the same type of partially organized firm brown thrombus. The combined weight of the lungs was 1,020 Gm.—a moderate

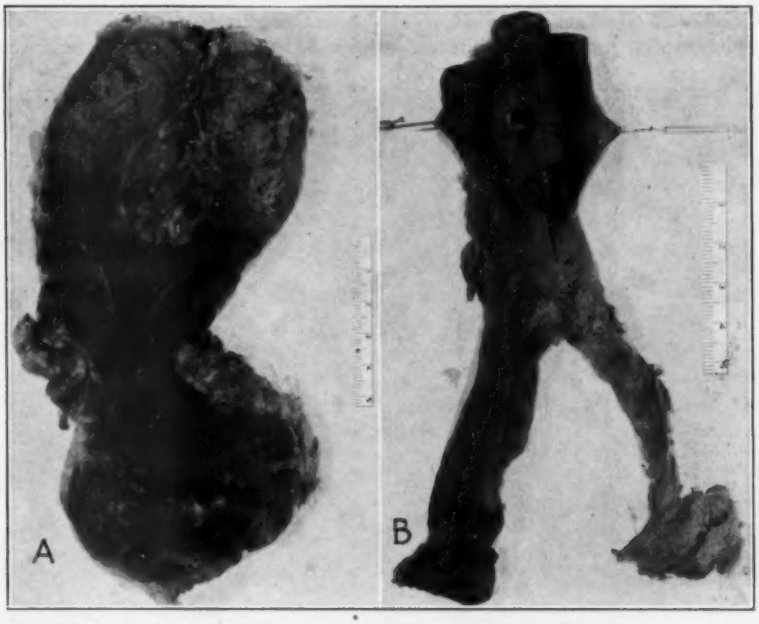

Fig. 1 (case 1).—*A*, the pancreas has been cut in a horizontal plane, and the two halves of the organ are shown attached at one margin. The tumor in the tail has completely replaced the normal racemose pancreatic tissue and is diffusely infiltrating the body. The irregular lighter areas on the cut surface are foci of necrosis. *B*, specimen of the inferior vena cava and the iliac veins. The vena cava is opened and is filled with a thrombus. The left femoral vein (shown on the right in the photograph) is smaller than the right. This is where the thrombus was removed at first operation.

increase. Section revealed numerous small areas of gray-white consolidation in the lower lobe of each lung, and pressure produced a small amount of slightly purulent material from the cut surfaces in these areas. Areas of deep black-red consolidation characteristic of infarcted pulmonary tissue were found in the upper and lower lobes of the left lung. These measured from 1 to 3 cm. in diameter. Several of the infarcted areas were surrounded by zones of lighter-colored tissue evidencing organization.

The weight of the brain was slightly increased, being 1,370 Gm. There was an area of recent softening in each occipitoparietal region. The tissue in these areas was not fluctuant, but it was notably softer than the surrounding substance. The area of softening in each hemisphere measured approximately 0.5 cm. in diameter. Nothing remarkable was noted in the examination of the cerebral

arteries or the leptomeninges. Step sections through the brain revealed nothing unusual except the areas of softening in each cerebral hemisphere previously noted. The softening extended through the centrum semiovale on each side to the most caudal part of the basal ganglions, but it did not involve these structures.

The several sections taken from the tumors in the tail of the pancreas, the liver and the parapancreatic lymph nodes showed essentially the same type of tumor.

In the sections taken from the pancreas there was complete replacement of normal parenchyma by large and small masses of tumor contained in a moderately abundant fibrous tissue stroma (fig. 2 *A*). The tumor was composed of well differentiated columnar epithelial cells which showed a tendency to form alveoli that in certain instances exhibited a striking resemblance to the normal alveoli

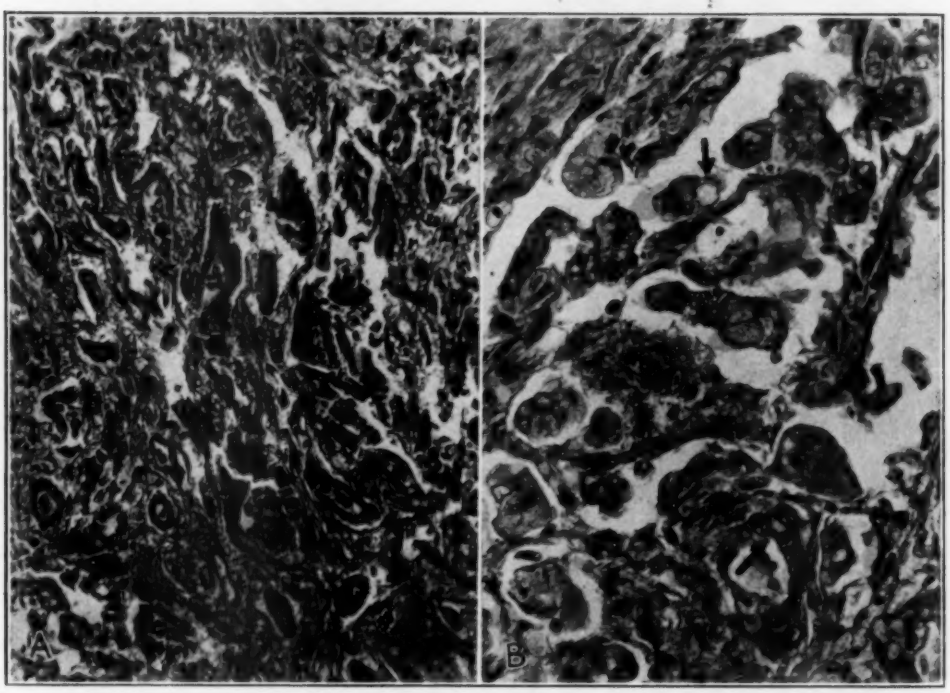

Fig. 2 (case 1).—*A*, tumor cell of columnar type forming well differentiated alveoli. Note the moderately abundant connective tissue stroma accompanying the tumor. Phloxine methylene blue stain; × 150. *B*, from the same section, with higher magnification. Tumor cells are seen in small groups and in some instances form small alveoli. Note granularity and vacuolization of the cytoplasm. The arrows indicate vacuoles in cytoplasm suggestive of goblet cells. Phloxine-methylene blue stain; × 475.

seen in the pancreatic tissue. No neoplastic giant cells were seen, but there were moderate numbers of mitotic figures and large areas of necrosis. Careful examination of the tumor cells disclosed that in many of the alveoli the columnar cells had a vacuolated cytoplasm, the vacuoles varying from a small amount of granularity of the cytoplasm to a single globule or vacuole remarkably similar to the cytoplasmic vacuolization of goblet cells of normal mucous membrane (fig. 2 *B*).

This type of vacuolization of the cytoplasm was not seen in all alveoli but occurred in approximately one fourth of the tumor cells. In some of the alveoli there appeared a small amount of granular-appearing substance. However, in no instance did the alveoli contain masses of material typical of mucus. In one of the sections a few neoplastic giant cells and large atypical mitotic figures were noted.

A section taken from the broad ligament revealed the ovarian and uterine veins filled with partially organized thrombi, with hemorrhagic infarction of the surrounding tissue and many large foci of hemorrhage. In some places there were complete destruction and necrosis of the wall of the vein, without evidence of primary inflammatory change in the wall. A section from the left iliac vein disclosed it to be filled with a well organized thrombus. The thrombus showed lines of Zahn and interlacing masses of fibrin containing disintegrated red

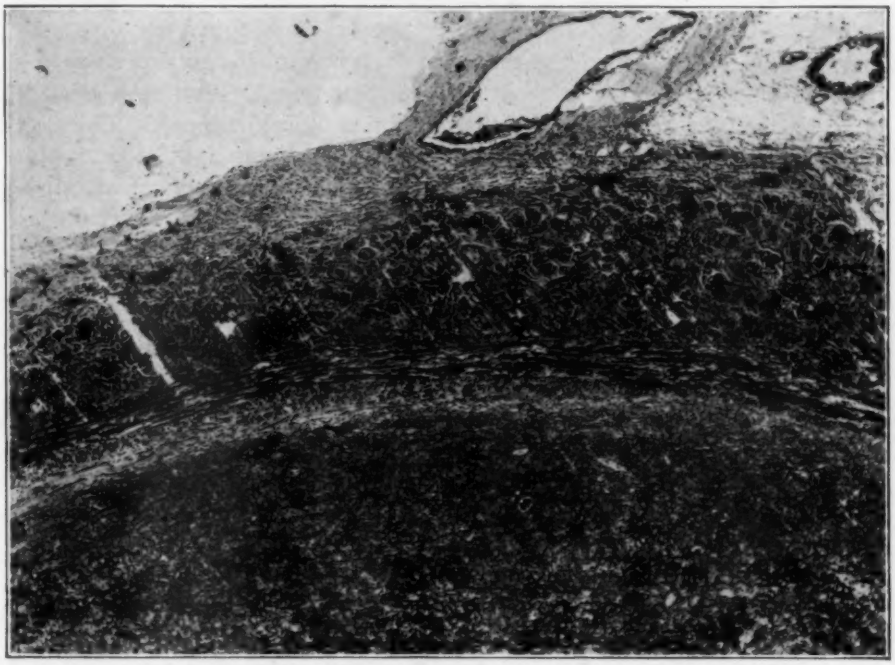

Fig. 3 (case 1).—Section of left iliac vein filled with organized thrombus. Note the absence of inflammatory disease or tumor in the wall of the vein and in the adventitia. Small capillaries and thin-walled blood spaces are seen in the thrombus shown in the lower part. Phloxine-methylene blue stain; × 40.

blood cells. A broad zone of organization with capillaries and thin-walled blood spaces was present, extending out from the wall for an estimated distance of a quarter of the diameter of the vein (fig. 3). Careful examination of the wall of the vein failed to present any evidence of primary inflammatory change in the muscularis, adventitia or intima of the vein.

Anatomic Diagnosis.—The anatomic diagnosis was well differentiated mucinous adenocarcinoma of the tail of the pancreas, metastatic carcinoma in the liver and parapancreatic lymph nodes, multiple partially organized thrombi in the inferior vena cava and in the iliac, femoral, ovarian and splenic veins, recent and partially organized infarcts in the lower lobe of the left lung and the upper

lobe of the left lung, recent encephalomalacia of the cerebral hemispheres and bronchopneumonia of the lower lobes of the lungs.

The diagnosis of mucinous adenocarcinoma was made only after the most careful histologic study of the tumor. This conclusion was based on finding many of the tumor cells with vacuolated and granular cytoplasm. A small amount of extracellular substance, presumed to be mucus, was noted within some of the alveoli. However, this substance was not entirely characteristic of the basophilic material produced by most mucus-producing tumor cells. The thrombi found in the systemic veins and in some of the radicles of the portal veins cannot be adequately explained as a stasis phenomenon since there was no demonstration of any obstruction by the tumor of the vena cava or the portal veins. Moreover, the initial complaint of the patient resulted from thrombosis of the veins in the legs some months before her death. The conclusion to be reached is that there was probably some disturbance in the coagulation mechanism of the blood to account for the thrombi observed. Unfortunately, no hematologic studies were made to investigate this possibility. The therapeutic administration of "dicumarol" was intended to prevent the further formation of thrombi and vitiated any study of the mechanism of the coagulation of the blood. The encephalomalacia noted in the cerebral hemispheres is best explained as a manifestation of thrombosis of small cerebral veins and part of the phenomenon of the altered coagulability of the blood.

CASE 2.—A 60 year old white woman entered the Santa Barbara Cottage Hospital on Feb. 27, 1945, complaining of severe pain in the upper part of the abdomen radiating to the back.

Present History.—For the past five months she had suffered from intermittent pain in the right upper quadrant of the abdomen. The pain frequently radiated to the back, without colic, was unrelated to intake of food, and occasionally was accompanied with nausea. Roentgenologic studies disclosed a defect involving the first part of the duodenum suggestive of ulcer, but a rigidly observed ulcer diet failed to provide relief from the pain.

Past History.—Symptoms of a rectal stricture had first developed twenty years before. Dilation of the anal canal provided only temporary relief until 1940, when a diagnosis of lymphopathia venereum was made and a permanent colostomy performed. After the operation the patient enjoyed good health until the onset of her present complaints.

Physical Examination.—Examination revealed a well developed but thin white woman in no immediate distress. The temperature was 98.2 F., the pulse rate 80 beats per minute, the respirations 20 per minute and the blood pressure 145 systolic and 95 diastolic. Examination of the head and neck disclosed nothing remarkable. The pulmonary fields were normal, and the only abnormality noted in the examination of the heart was a faint systolic murmur heard over the precordium. Examination of the abdomen revealed two colostomy openings, the opening of the proximal loop being situated to the left of the umbilicus and slightly below it, while the stoma of the distal loop was implanted in the midline approximately 5 cm. above the symphysis pubis. The pelvic examination was noncontributory.

The anorectal canal was obliterated by fibrous tissue. The extremities were not remarkable and were free from edema.

Roentgenologic Examination.—A roentgenogram made after a barium meal revealed a normal stomach. The duodenal bulb was constantly deformed and moderately irritable. Fluoroscopically, a crater was demonstrated. This was also noted in the subsequent roentgenograms.

Laboratory Examination.—The erythrocyte count on the patient's admission to the hospital was 4,460,000 and the hemoglobin content 10.5 Gm. per hundred cubic centimeters. The white blood cell count was 7,700. Examination of the urine disclosed albumin (3 plus) but no sugar and nothing remarkable in the sediment. A gastric analysis showed a total acidity of 19 degrees and no free hydrochloric acid. The icteric index was 4.3. The reactions to the Kahn and Kolmer serologic tests for syphilis were negative, as were the reactions to cutaneous sensitivity tests with the Frei antigen. The presence of occult blood (2 plus), without other significant findings, was reported after one examination of the stool.

Clinical Course.—A ten day regimen for the treatment of ulcer resulted in no amelioration of the symptoms. Opiates were required for relief of pain, and there were frequent vomiting episodes. At this time the attending physician expressed the opinion that pancreatitis complicated the peptic ulcer and suggested an exploration of the upper part of the abdomen. This opinion was concurred in by the surgical consultant, and the patient was prepared for operation. The lower end of the stomach and the proximal part of the duodenum were found adherent to a tumor mass, which was palpated in the body of the pancreas. There was a traction deformity near the outlet of the stomach, but no crater was demonstrated. Several firm nodules of tumor were noted in the mesentery below the pylorus. A gray-white nodule 2.5 cm. in diameter was seen on the superior surface of the right lobe of the liver, near the dome. The patient left the hospital on the twenty-first postoperative day with her general condition unimproved except that she required only occasional hypodermic injections of codeine for discomfort and pain.

Second Admission to the Hospital.—The patient was readmitted to the Santa Barbara Cottage Hospital on July 27, 1945, seven weeks after her discharge. While at home, the pain in the epigastrium and right upper abdominal area had become progressively more severe, and opiates were needed at shorter intervals each day.

Course in the Hospital.—Physical examination disclosed little change except for moderate edema of both ankles and feet. There was a new complaint of dyspnea. These new developments were attributed to beginning cardiac decompensation. Three grains of codeine (0.19 Gm.) was administered by hypodermic injection as often as every two hours. The course was steadily downhill, and the patient died on the sixteenth day in the hospital.

Observations at Necropsy.—Permission was granted for the examination of the thoracic and abdominal organs. The significant observations were as follows:

A healed scar was noted in the upper part of the mid-abdomen, and there were two functioning colostomy openings in the lower part of the mid-abdomen approximately 8 cm. apart. The examination of the lower extremities disclosed a moderate amount of pitting edema of the subcutaneous tissue. The peritoneal cavity contained 1,000 cc. of dark amber fluid. All the peritoneal surfaces were covered with numerous raised semitranslucent light gray nodules of tumor. The nodules in many instances formed adhesions between the loops of small intestine.

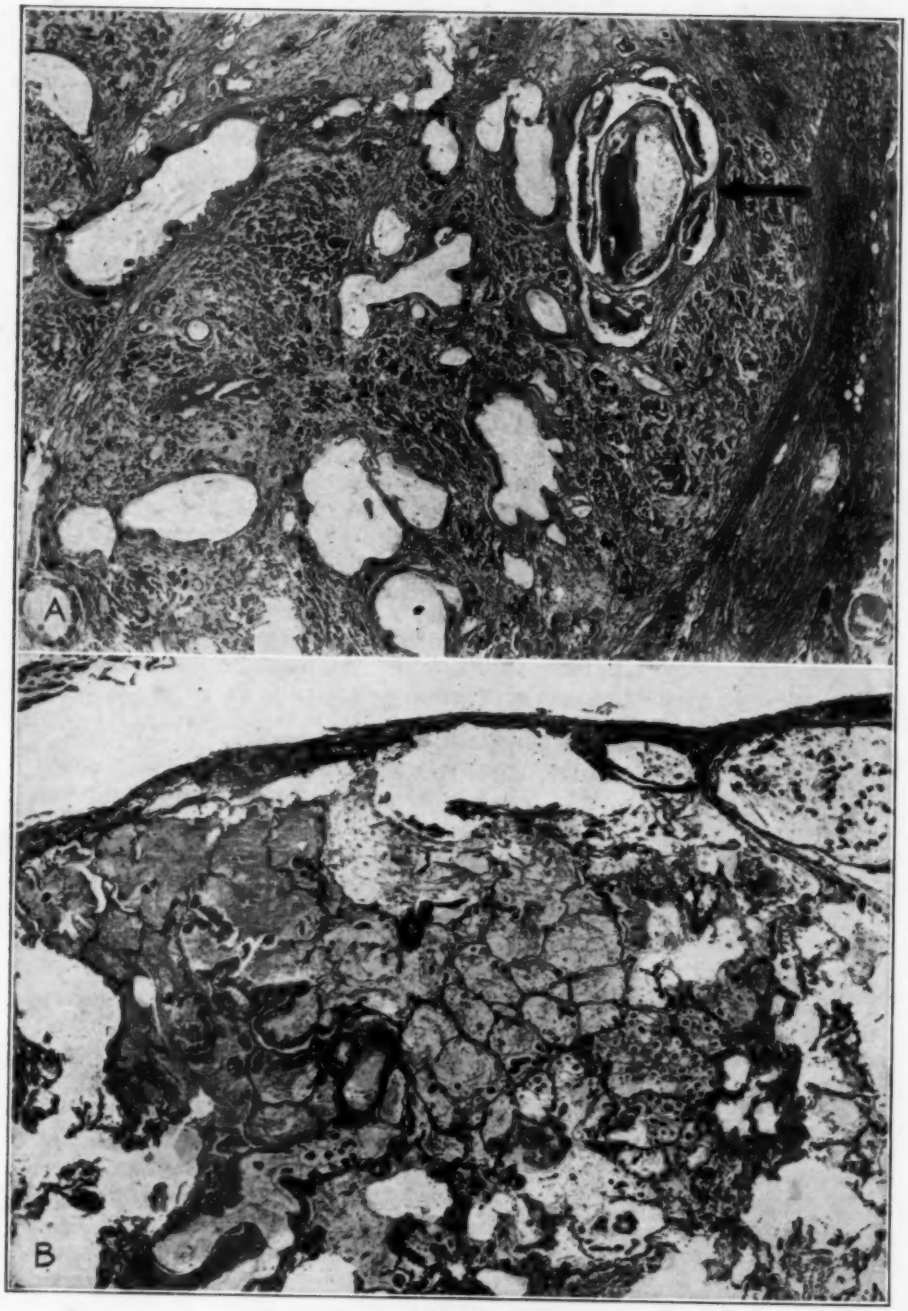

Figure 4

(See legend on opposite page)

A firm mass was palpated in the body of the pancreas. Large masses of firm gray-white semitranslucent tumor were found in the para-aortic lymph nodes.

The pancreas weighed 180 Gm. and was covered by scattered discrete small foci of tumor. External examination of the organ revealed a 6 cm. mass of finely granular, firm gray tumor replacing the body and a large part of the tail. The tumor merged imperceptibly with and diffusely infiltrated the head of the organ and the remaining part of the tail. There was only 2 cm. of the head of the pancreas and only 4 cm. of the tail uninvolved by the tumor. Section of the pancreas in a transverse horizontal plane disclosed a semitranslucent gray-white tissue that had a mucoid-appearing surface replacing the normal racemose tissue.

The weight of the lungs was markedly increased, the combined weight being 1,500 Gm. Small masses of semitranslucent, gray and dark red tumor were noted on the surface, varying from a few millimeters to 1.5 cm. in diameter. Section revealed the same type of tissue scattered in all the lobes. The nodules were most numerous in the lower lobes, and in these areas there was nearly complete consolidation of the lung by the tumor. The same type of tumor was noted in the mediastinal and peribronchial lymph nodes.

The weight of the liver was moderately increased, being 2,000 Gm. The capsule was covered with numerous raised masses of translucent pink tumor similar to those previously described on the peritoneal surfaces of the small and large intestines. Section revealed masses of tumor scattered through the liver and measuring from a few millimeters to 2 cm. in diameter. There was a notable narrowing of the lumen of the rectum, beginning 1 cm. from the anal orifice, and 8 cm. from the anal orifice there was complete obliteration of the lumen. In the narrowed part the wall was heavily infiltrated with large amounts of fibrous tissue. The muscularis in this area appeared unusually prominent, measuring in some places up to 7 mm. There was no significant pathologic change in the remaining part of the colon from the rectum to the colostomy. Examination of the principal radicles of the portal veins and the vena cava disclosed the following: In the inferior vena cava and in the iliac, hypogastric and colic veins there were masses of mottled brown and red thrombi that showed varying degrees of organization and adherence to the wall. The thrombus in the inferior vena cava extended from 8 cm. above the bifurcation down into the iliac veins. There was a continuous mass of thrombus extending from the iliac veins into the hypogastric veins. The inferior colic veins were filled with the same type of brown-red thrombi, and the thrombi extended to within 10 cm. of the point of entrance of the inferior colic veins into the portal vein.

Microscopic examination of the sections taken from the tumor in the pancreas showed essentially the same type of tumor. There was complete replacement of all the glandular elements of the pancreas by masses of tumor cells contained in an abundant fibrous tissue stroma. The tumor formed large and small alveoli that in many instances produced large cystic spaces (fig. 4*A*). The tumor

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Fig. 4 (case 2).—*A*, pancreas invaded by tumor. The tumor is seen as large, well differentiated alveoli in all parts of the field infiltrating the pancreatic parenchyma. The tumor alveoli are filled with a basophilic-staining mucus. Note the invasion of the perineuronal lymphatic spaces of a medium-sized nerve trunk marked by an arrow in the upper right part of the field. Phloxine methylene blue stain; × 60. *B*, section of lung showing tumor nodule. Alveoli are filled with basophilic mucus and small and large clusters of tumor cells, some forming an epithelial lining for the alveolus. Note the subpleural lymphatic vessel in the upper right area of the field filled with tumor. Phloxine-methylene blue stain; × 60.

cells showed a remarkable tendency to produce a slightly reticulated-appearing, basophilic-staining mucus that accumulated in the lumens of the alveoli. The cells lining the alveoli were tall columnar cells in which the cytoplasm frequently contained large vacuoles of basophilic-staining substance. The tumor demonstrated an unmistakable tendency to invade the perineural lymphatic spaces (fig. 4 *A*). A small piece of pancreatic parenchyma seen on one side of the section revealed advanced acinar atrophy.

The same type of tumor as was noted in the sections from the pancreas was observed in the liver. A section of lung containing tumor revealed the alveoli to be filled with masses of basophilic-staining substance surrounding small clusters of tumor cells. In some of the alveoli the tumor lined the alveolar space. Subpleural lymphatic spaces were filled with the tumor (fig. 4 *B*).

Two sections taken from the rectal stricture showed essentially the same type of pathologic change. In one of the sections the surface was covered with an atrophic type of stratified squamous epithelium, while in the second section there was no epithelium but a poorly preserved layer of connective tissue containing a few capillaries with small foci of lymphocytes and plasma cells. The wall was infiltrated with connective tissue and scattered foci of lymphocytes and plasma cells. Sections from the hypogastric veins and the portal veins containing the thrombi disclosed partial organization of the thrombi but no tumor or inflammatory change in the wall or in the adventitial tissues.

Anatomic Diagnosis.—The anatomic diagnosis was mucinous adenocarcinoma of the body of the pancreas, metastatic mucinous carcinoma in the lungs, liver and peritoneum, partially organized thrombi in the inferior vena cava and the iliac, hypogastric and colic veins, edema of the subcutaneous tissues of the lower part of the legs, ascites and stricture of the rectum.

The mucinous carcinoma observed in this case, a well differentiated tumor producing large amounts of mucus, was not unusual in any respect. Tumors of this histologic type are frequently found arising in other parenchymatous organs and in the head as well as in the body and tail of the pancreas. Explanation of the partially organized thrombi in the inferior vena cava and in the iliac, hypogastric and colic veins strongly points to some altered coagulability of the blood rather than to a mechanical factor or primary change in the wall of the vein. Gross examination of the vena cava and of the portal veins failed to demonstrate any obstruction above the thrombi. Moreover, the occurrence of thrombi on both systemic and portal sides of the venous circulation is additional evidence against a mechanical factor causing the thrombosis. Unfortunately, the laboratory work did not include studies of the coagulability of the blood.

COMMENT

Although the clinical histories of the 2 patients discussed have little in common, the pathologic study in each case demonstrated remarkably similar pathologic changes. In each there was found a carcinoma of the tail or body of the pancreas in which the tumor cells were producing intracellular and extracellular substance. One of the tumors was producing an abundant amount of basophilic-staining mucus, while in the other only a small amount of intracellular and extracellular

substance was demonstrated. There was an associated phlebothrombosis involving both the portal and the systemic venous systems in each case. The most reasonable explanation for the phlebothrombosis was some alteration of the blood-clotting mechanism, since such factors as debility, cardiac decompensation, mechanical pressure, invasion of the venous wall by tumor, primary inflammatory change in the wall of the vein and disturbed hepatic function could be excluded.

These 2 cases, therefore, are examples of a characteristic syndrome, first described by Sproul,[6] in which a high incidence of phlebothrombosis is associated with carcinomas arising in the tail or the body of the pancreas. The tumor in each of the cases was composed of cells producing an intracellular substance, an observation giving support to Kenney's[7] suggestion that the substance produced by the cells is responsible for the altered blood clotting. It must be pointed out, however, that this is only inferential evidence, since no specific substance has been identified in the neoplastic tissue. The existence of this syndrome is well evidenced by the studies of Sproul, who found an incidence of 31.3 per cent of disseminated phlebothrombosis in carcinomas of the tail and body of the pancreas in 4,258 consecutive autopsies, and by the work of Kenney,[7] who reported 21 cases of this type of cancer, with an incidence of 33.3 per cent of multiple venous thrombi, in 9,800 autopsies. Although carcinoma of the tail and body of the pancreas is associated with an impressive incidence of multiple phlebothrombosis, this condition is occasionally seen with mucinous-producing carcinoma which is primary in other organs or with carcinoma of the head of the pancreas. We have recently observed it with the former, and Sproul[6] has reported a 1.37 per cent incidence of disseminated phlebothrombosis in carcinoma of the stomach.

Further knowledge of this problem will depend on studies of the blood-clotting mechanism during the life of patients suspected of having the syndrome, which should include fibrinogen and prothrombin tests and determination of the platelets. Detailed pathologic and chemical studies of the neoplastic tissue obtained at autopsy from such patients might give information about the presence or absence of a specific substance responsible for the altered blood clotting. An investigation should rule out known facts which might influence the coagulability of blood, such as stimulation of the bone marrow from hemorrhage, increased production of platelets, increased intake of vitamin K and the presence of some toxic substance in the blood that would act directly on fibrinogen to produce fibrin.

Specific knowledge concerning the relationship of disturbance in the mechanism of blood coagulation to the syndrome under consideration might prove helpful in the establishment of an early diagnosis of the disease. To date, no clearcut clinical differentiation has been made between multiple thrombophlebitis ("migrating phlebitis") and

multiple phlebothrombosis. Once these two conditions can be established as separate clinical entities, it is possible that a definite clue may be had to the existence of mucinous carcinoma situated in the pancreas, stomach or lung, the odds greatly favoring the first-mentioned organ. Such evidence may well constitute an indication for abdominal exploration and possible cure of the disease.

Available evidence does not give definite information concerning the time of appearance of the thrombosis in relationship to the development of the tumor in the pancreas. One of Kenney's [7] patients had signs of venous thrombosis antedating by three months any symptoms which might have been attributed to systemic or visceral disease. In the first case reported in this communication the patient's initial complaint was that of pain and swelling in the leg, and not until late in the course of her illness was there evidence of disease which could not be explained entirely on the basis of the peripheral vascular disturbance; however, only three months had transpired between the apparent onset of venous thrombosis and death from advanced carcinoma of the pancreas. One logically concludes that in this case the tumor had metastasized before the first symptoms of vascular thrombosis were observed. Until a larger number of cases of this syndrome have been adequately studied, the relative time of the appearance of the symptoms and signs of phlebothrombosis in the course of mucinous adenocarcinoma is a point for conjecture. It is of interest that in the most recent published papers on venous thrombosis [8] no mention was made of this syndrome.

SUMMARY

Two cases of carcinoma of the pancreas, in 1 of which the primary tumor was in the tail and in the other in the body, are reported together with the observations at autopsy. In each patient multiple venous thrombi occurred in both the portal and the systemic venous circulations, and in each the tumor cells were producing an intracellular and an extracellular substance. Carcinomas of the body and tail of the pancreas in which the cells produce both intracellular and extracellular substances are frequently associated with multiple venous thrombi, a circumstance that is now recognized as indicative of a definte clinical and pathologic syndrome.

Nothing is known concerning the disturbance in the blood-clotting mechanism which is responsible for the thrombosis. In all cases of disseminated phlebothrombosis attention should be directed to the possible existence of carcinoma and a careful search made for signs and symptoms pointing to its identification.

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8. Aycock, T. B., and Hendricks, J. W.: Problems of Venous Thrombosis, J. A. M. A. **133**:1258-1263 (April 26) 1947. Allen, A. W.; Linton, R., and Donaldson, G. A.: Venous Thrombosis and Pulmonary Embolism, J. A. M. A. **133**:1268-1276 (April 26) 1947.

SURGICAL TREATMENT OF CARCINOMA OF THE AMPULLA OF VATER AND THE EXTRAHEPATIC BILE DUCTS

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THE TECHNIC for partial duodenopancreatectomy described in 1935 by Whipple and his associates [1] has given impetus and encouragement for the radical treatment of tumors arising in a region previously considered inaccessible. Prior to this time, attempts had been made to resect ampullary tumors by a transduodenal approach; however, the results were by no means satisfactory, and the complications which arose in operating on jaundiced patients usually did not justify the procedures. It is hardly remarkable that before the advent of methods for controlling the tendency toward bleeding in the presence of jaundice palliative operations were usually performed. The use of vitamin K, a better understanding of the function of the liver and better preoperative preparation and postoperative management in conjunction with improved technical procedures have made possible wide excision of carcinoma of this region.

During the past eleven years, some modifications of the original two stage operation as performed by Whipple have been made. A number of methods [2] have been described for partial duodenopancreatectomy. Variations have been suggested in the method by which the biliary flow is diverted to the gastrointestinal tract as well as in the type of gastroenterostomy done. Various opinions have been

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1. Whipple, A. O.; Parsons, W. B., and Mullins, C. R.: Ann. Surg. **102**:763-779, 1935.

2. (*a*) Brunschwig, A.: Surg., Gynec. & Obst. **65**:681-684, 1937. (*b*) Trimble, I. R.; Parsons, J. W., and Sherman, C. P.: ibid. **73**:711-722, 1941. (*c*) Moreland, R. B., and Freeman, B. S.: Surgery **9**:712-719, 1941. (*d*) Hunt, V. C.: Ann. Surg. **114**:570-602, 1941. (*e*) Dennis, C.: Surgery **12**:201-206, 1942. (*f*) Cattell, R. B.: S. Clin. North America **23**:753-766, 1943. (*g*) McCall, C. H., and Waugh, J. M.: Proc. Staff Meet., Mayo Clin. **19**:147-152, 1944. (*h*) Orr, T. G., and Walker, G. A.: Surg., Gynec. & Obst. **80**:149-151, 1945. (*i*) Behrend, M.: Am. J. Surg. **72**:599-603, 1946.

expressed in regard to the method of handling the resected surface of the pancreas including the duct of Wirsung. Whipple and his associates (as well as others) have stated that ligation of the main pancreatic duct does not lead to complications or sequelae. Other observers [3] have apparently believed that the external pancreatic secretion is an aid in digestive physiology although it may not be essential to life. From a technical standpoint it is possible to divert the pancreatic secretion to the gastrointestinal tract by either end to end pancreaticojejunostomy, end to side pancreaticojejunostomy or the implanting of the duct of Wirsung directly into the jejunum with or without the use of a cannula. Since the flow of pancreatic secretion can be reestablished in the gastrointestinal tract, the added surgical effort to accomplish this end seems to be desirable.

TABLE 1.—*Chronologic Occurrence of Carcinoma of the Ampulla of Vater and the Extrahepatic Bile Ducts*

Year	Carcinoma of Ampulla of Vater	Carcinoma of Extrahepatic Bile Ducts
1937	..	..
1938	..	..
1939	1	1
1940	2	2
1941	1	3
1942	3	1
1943	..	..
1944	1	1
1945	..	1
1946	..	1
10 years	8	10
Clinical incidence	0.027%	0.056%

The problem of treatment of carcinoma of the ampulla of Vater and the extrahepatic bile ducts is perhaps more serious than that of treatment of cancer elsewhere in the body because the diagnosis is usually made late in the growth of the neoplasm and in many instances treatment is delayed in an attempt to make a correct differential diagnosis.

In the past ten years we have been particularly interested in this disease among patients at the Cincinnati General Hospital. This report is based on the analysis of cases in which the patients were proved to have carcinoma of the ampulla of Vater or of the extrahepatic bile ducts by operation and biopsy, operation and autopsy or autopsy alone. We are advertently excluding carcinoma of the head of the pancreas in this study. In the period 1937 to 1946 inclusive (table 1) we have treated 8 patients with carcinoma of the ampulla of Vater and 10 patients with carcinoma of the extrahepatic bile ducts. In the final analysis, the diagnosis and treatment may be considered the same for the two condi-

3. Boyce, F. F.: Surgery **4**:51-63, 1938. Varco, R. L.: ibid. **18**:569-573, 1945.

tions. For the purposes of clarification and analysis, however, we are presenting this discussion by separate consideration of these two regions.

CARCINOMA OF THE AMPULLA OF VATER

Incidence.—The occurrence of carcinoma of the ampulla of Vater is relatively rare. Lieber, Stewart and Lund [4] reported 14 cases among 22,152 necropsies at the Philadelphia General Hospital between 1920 and 1936 and 4 cases at the Jefferson Hospital, Philadelphia, among 4,154 necropsies. This is an incidence of 1.13 per cent of all carcinomas. In our study of 8 cases observed at the Cincinnati General Hospital between 1937 and 1946, during which time 31,935 patients were admitted to the surgical service, a clinical incidence of 0.027 per cent was found.

Generally the age incidence parallels that in cancer of the alimentary tract. Most patients are over 50 years of age, but the condition is

TABLE 2.—*Carcinoma of the Ampulla of Vater Showing Distribution of Age, Sex and Race*

Case	Age	Race		Sex	
		White	Negro	Male	Female
1	62	+	..	+	..
2	38	+	..	+	..
3	68	+	..	+	..
4	67	+	..	..	+
5	67	+	..	..	+
6	48	..	+	+	..
7	72	+	..	+	..
8	39	+	..	+	..
	57.2	7	1	6	2

occasionally seen in younger persons. Males are affected more often than females, the ratio being about 2 to 1.

In our own series (table 2) the average age was 57.2 years. The oldest patient was 72 years, and 3 patients were under 50, being 38, 39 and 49 years respectively. Of the 8 patients, 7 were white and 1 was a Negro. Six were men, and 2 were women, affording a ratio of 3 to 1.

Pathologic Process.—Brunschwig [5] pointed out that the terms "ampulla of Vater" and "papilla of Vater" have been used indiscriminately in the literature. From a practical viewpoint and because common usage has firmly established "ampullar carcinoma" as including all peripapillary carcinomas, there is no real objection to this terminology.

4. Lieber, M. M.; Stewart, H. L., and Lund, H.: Ann. Surg. **109**:219-245 and 383-429, 1939.

5. Brunschwig, A.: The Surgery of Pancreatic Tumors, St. Louis, C. V. Mosby Company, 1942.

Grossly, there are two types of ampullary carcinoma, namely, the papillary type and the ulcerating type. At the time of operation, palpation of the ampullary region may be difficult because of preexisting pancreatitis. Hence the discovery of the true pathologic state may require direct visualization by duodenotomy. On rare occasions stones lodged in the ampulla have made the exact diagnosis difficult.

Histologically, the tumors range from typical adenocarcinoma with papillary cystic structure to configurations of solid masses or cords of malignant cells representing scirrhous carcinoma.

Neoplasms in this location spread to the duodenal wall and the pancreas and into the common bile duct by direct extension. It is thought that metastasis to the regional lymph nodes, the liver and the pulmonary structures is by the lymphogenous route and also by the portal vein. There is a general impression that carcinomas of this region are relatively low grade tumors and that the metastases occur late. Our clinical interpretation and pathologic observations have led us to conclude that such an argument is not valid. It is now known that obstruction of the bile flow, giving rise to hepatic damage with its subsequent complications, leads to an early death in many cases. Further, one of us has observed gross metastases to the liver developing in a seventeen day interim between the stages of a Whipple procedure.

Signs and Symptoms.—Pain is a characteristic symptom of this disease. Sharpe and Comfort [6] reported pain of some type in 60 per cent of their cases, and others believe the incidence to be even higher. There is no characteristic pain associated with carcinoma of the ampulla of Vater. Usually there is epigastric discomfort, described by some patients as being a dull, boring or sharp pain, either intermittent or continuous and not associated with the ingestion of food. It may or may not radiate to the various areas of the upper part of the abdomen. In many instances it radiates through the body to the back. It would seem desirable to condemn the older ideas about "painless jaundice" and to be more exacting in "history taking," particularly in teaching younger physicians the concepts of early diagnosis of this lesion.

Anorexia and loss of weight may overshadow all other symptoms. Nausea, vomiting and diarrhea may or may not accompany these symptoms. Chills and fever as described by Charcot usually suggest cholangitis, which may exist because of obstruction by the tumor, an associated condition of stones in the common duct or preexisting infection of the extrahepatic biliary tract.

Jaundice is usually observed in the course of this disease, and variations in its degree indicate that no characteristic clinical type exists. Three types may occur, namely, painless intermittent jaundice, painless constant jaundice and painful intermittent jaundice.

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6. Sharpe, W. S., and Comfort, M. W.: Am. J. M. Sc. **202**:238-245, 1941.

Palpation of a distended gallbladder in the presence of established obstructive jaundice suggests that the obstruction is neoplastic in nature. This is a confirmation of Courvoisier's sign. Hepatic enlargement is said to occur in 86 per cent of cases.

Laboratory Features.—Anemia of a secondary variety may be observed in a majority of cases.

The persistence of occult blood in the stool cannot be overemphasized. Cooper [7] has said, "This is one distinctive feature about this disease; the only one not regularly encountered in obstructive jaundice due to other causes. As a criterion it is not without its pitfalls, however, for all cases of long standing jaundice may have cholemic hemorrhages." Halsted [8] reported fatal hemorrhage in a case of carcinoma of the ampulla of Vater, and Cooper reported it in 2 of the 14 cases which he reviewed. Daily examination of the stools for occult blood are imperative as an aid in making a diagnosis.

Duodenal drainage may also be helpful in arriving at an accurate diagnosis, because the presence of bloody duodenal mucus is suggestive of ulcerating ampullary carcinoma. When other findings point toward the presence of a neoplasm in complete obstructive jaundice, the absence of bile in the duodenal drainage may be indicative of a malignant process in this region.

Roentgenologic studies may be helpful in confirming a diagnosis. In some instances, careful fluoroscopic studies reveal a "filling defect" in the region of the papilla of Vater. Flattening or displacement of the second portion of the duodenum may suggest carcinoma of this area. In the early stage of the growth of a tumor in the periampullary region associated with edema the "reversed 3 sign" may be considered almost pathognomonic of the lesion, as has been pointed out by Frostberg.[9]

Differential Diagnosis.—Most observers now agree that a positive diagnosis can be made only by exploration. The main problems in differential diagnosis are stones in the common duct and carcinoma of the head of the pancreas, of the duodenum or of the extrahepatic bile ducts.

A summary of the important findings which lead to a working clinical diagnosis of carcinoma of the ampulla of Vater are: (1) history of persistent epigastric discomfort or pain; (2) history of anorexia and loss of weight; (3) clinical substantiation of obstructive jaundice; (4) anemia; (5) persistent occult blood in the stool; (6) presence of blood in the duodenal secretion, and (7) roentgenologic evidence of a "filling defect" or a "reversed 3 sign" in the periampullary region.

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7. Cooper, W. A.: Ann. Surg. **106**:1009-1035, 1937.
8. Halsted, W. S.: Boston M. & S. J. **141**:645-654, 1899.
9. Frostberg, N.: Acta radiol. **19**:164-173, 1938.

Treatment.—From recent surveys [10] by others and from our own observations we conclude that early diagnosis and early surgical treatment can be the only logical approach in dealing with this lesion. For patients not operated on, death from damage to the liver (which ultimately is related to all basic human physiology) is certain. The average life expectancy for the untreated patient after the onset of symptoms varies between three and eight months.

When an early diagnosis is made, the treatment of choice is a partial duodenopancreatectomy, with reestablishment of normal function so far as is surgically possible. Whether the operation is done in a one or in a two stage procedure must be left to the judgment of the surgeon in the individual case.

Analysis of Cases and Results (*table 3*).—Eight cases of proved carcinoma of the ampulla of Vater are reported. The operability rate for radical partial duodenopancreatectomy was 50 per cent. A palliative operation was definitely planned at the time of exploration in 3 cases; the procedures in these included cholecystogastrostomy and choledochoduodenostomy. In 1 instance (case 7) a two stage Whipple procedure was planned; however, the patient died from complications of his obstructive jaundice before resection could be performed. For this reason we have considered the operation as being a palliative one.

Of the 4 patients on whom partial duodenopancreatectomy was performed, 3 have died and 1 is living at the time of this report. This gives a comprehensive mortality rate of 87.5 per cent for the ten year period.

It should be noted, however, that the patient in case 1 lived seven years and three days. He recently died from obstructive jaundice due to metastatic melanosarcoma of the liver and gallbladder which occluded the common duct at the junction of the cystic duct. As far as can be ascertained, this represents the longest survival period for any patient who has submitted to this radical procedure.

The patient in case 2 had a two stage partial duodenopancreatectomy and survived for only twenty-eight days, death coming as a result of intestinal obstruction and cardiac failure.

Case 6 represents an instance in which a one stage partial duodenopancreatectomy was done. The patient lived for five months and five days and died at home. The exact cause of death was not determined; however, it may be presumed that his diseased liver was a factor in causing his death.

The patient in case 8 submitted to a two stage partial duodenopancreatectomy. This is an instance in which a pancreaticojejunostomy

10. Whipple, A. O.: Ann. Surg. **114**:612-615, 1941; New England J. Med. **226**:515-526, 1942. Child, C. G., III.: Ann. Surg. **119**:845-855, 1944. Cattell,[2f]

TABLE 3.—*Carcinoma of the Ampulla of Vater*

Case	Age, Yr.	Race	Sex	Date of Admission	Operative Procedure *		Result		Period of Survival			Comment
					Duodeno-pancreatectomy	Palliative Procedure	Living	Died	Yr.	Mo.	Days	
1	62	White	Male	10/18/39	Two stage Whipple (a) 10/31/39 (b) 12/ 9/39	.....	.....	12/12/46 (Survived for 7 years and 3 days)	7	..	3	Melanoma of skin; metastases to liver, gallbladder and duct system; carcinoma of prostate; died of obstructive jaundice and cardiac failure; autopsy
2	38	White	Male	4/12/40	Two stage Whipple (a) 4/25/40 (b) 5/17/40	.....	.....	6/14/40	..	..	28	Died of intestinal obstruction and cardiac failure; no autopsy
3	68	White	Male	10/22/40	.....	Cholecysto-gastrostomy 11/12/40	.....	11/15/40	..	..	3	Died from obstructive jaundice and cardiac failure; autopsy
4	67	White	Female	6/12/41	.....	Cholecysto-gastrostomy 6/24/41	.....	10/19/41	..	4	7	Diabetes mellitus; no autopsy
5	67	White	Female	1/ 8/42	.....	Choledocho-duodenostomy and cholecys-tostomy 1/24/42	.....	10/ 4/42	..	8	11	No autopsy
6	48	Negro	Male	5/27/42	One stage Whipple 6/12/42	.....	.....	11/17/42	..	5	5	Cholangitis; hydrohepatosis; metastatic carcinoma of liver; autopsy
7	72	White	Male	11/20/42	.....	Cholecystoje-junostomy and jejunoje-junostomy 12/5/42	.....	12/18/42	..	..	13	Two stage Whipple operation planned; patient died of complications of obstructive jaundice before second operation could be done; autopsy
8	39	White	Male	3/24/44	Two stage Whipple (a) 4/ 8/44 (b) 4/29/44	.....	2 yr. and 10 mo.†					
Total				8 cases	4	4	1	7				

* The operability rate was 50 per cent for duodenopancreatectomy and 50 per cent for palliative procedures. The mortality rate (ten year period) was 87.5 per cent.

† Patient still living, approximately 2 years 10 months later.

was done. The patient is living at the time of the writing of this report, approximately two years and ten months after radical resection of the tumor.

The technical details for partial duodenopancreatectomy have been well outlined elsewhere, and repetition here is not indicated. We believe, however, that 2 of our cases are of sufficient interest to be recorded in some detail.

Case 1 (table 3).—A white male laborer was 62 years old at the time of his admission to the Cincinnati General Hospital on Oct. 18, 1939. He complained of abdominal pain of three months' duration accompanied with belching, diarrhea, weakness, loss of weight and jaundice. There was no nausea or vomiting.

On his admission he appeared robust, and there was moderate cardiac enlargement. The blood pressure was 210 systolic and 110 diastolic. He was deeply jaundiced. The liver was palpable several centimeters below the right costal

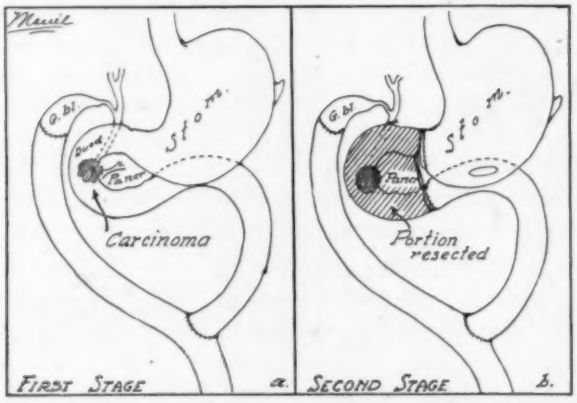

Fig. 1 (case 1, table 3).—*a*, first stage Whipple operation, showing end to end cholecystojejunostomy and end to side jejunojejunostomy; *b*, second stage Whipple operation, showing partial duodenopancreatectomy (shaded area) and side to side posterior gastroenterostomy.

margin, and below it was a rounded mass about 3 cm. in diameter, which was thought to be an enlarged gallbladder.

Laboratory studies gave the following significant results: The blood count was essentially normal. The reaction to the Kahn test was negative, and the icteric index was 115. The urine gave a negative reaction for urobilin and a positive reaction for bile. The stool was clay-colored and gave a 2 plus reaction to the guaiac and benzidine tests. The prothrombin time was 100 per cent normal. The blood phosphatase was 16.8 Bodansky units and the blood cholesterol 400 mg. per hundred cubic centimeters. The jaundice increased while the patient was under observation. A diagnosis was made of complete obstructive jaundice probably due to neoplasm.

On October 31 an exploratory laparotomy was performed, and a cylindric mass arising in the ampulla of Vater was discovered. It was decided to carry out a partial duodenopancreatectomy as described by Whipple. The first stage was carried out at this time (fig. 1 *a*). This consisted of dividing the jejunum about 2 feet (61 cm.) below the ligament of Treitz. The distal end was mobilized to accomplish

an end to end cholecystojejunostomy and the proximal end used to make an end to side jejunojejunostomy into the distal limb about 12 to 14 inches (30 to 35 cm.) below the anastomosis with the gallbladder. Both of these anastomoses were made aseptically over Stone clamps.

The patient did well postoperatively. The icteric index dropped rapidly, and the stools became normal in color. On November 24 the icteric index was 17. Roentgenograms showed no evidence of regurgitation of barium into the gallbladder.

On December 9 the second stage of the operation was carried out (fig. 1 *b*). First a gastroenterostomy was performed; then the pyloric end of the stomach was divided and the proximal end closed. The third portion of the duodenum was divided and the distal end closed. The duodenal loop, the head of the pancreas and the lower end of the common bile duct were then removed in one block (fig. 2). The cut end of the pancreas was closed with interrupted silk sutures and the com-

Fig. 2.—Drawing of gross specimen, showing the adenocarcinoma of the ampulla of Vater.

mon duct ligated with braided silk. Drains were placed to the retroperitoneal area of resection, and the abdomen was closed.

Examinations of the specimen showed an adenocarcinoma (fig. 3) of rather low grade malignancy arising from the ampulla of Vater. No metastases were found in any of the lymph nodes removed.

The patient did well for about one week, when profuse drainage of bile and pancreatic secretion occurred from the wound. This continued for some time, and injection of iodized poppyseed oil 40 per cent into the sinus showed that it communicated with the dilated bile duct system. After some weeks the bile fistula closed, but the pancreatic fluid continued to drain. Fatty, foamy stools developed, with considerable diarrhea, which was relieved by intake of raw pancreas and pancreatin by mouth. The pancreatic fistula persisted for many months, closing from time to time. Each time it closed, however, chills and fever would

develop. The patient was readmitted to the hospital five times in 1940 for dilatation of the fistulous tract. At one time, injection of iodized poppyseed oil 40 per cent outlined dilated ducts in the pancreas. During these months he remained weak and diarrhea was persistent though lessened by intermittent pancreatic replacement therapy. It was found impossible to keep his blood count and plasma proteins at normal levels.

After consideration it was decided to implant the pancreatic fistula into the gastrointestinal tract. On May 1, 1941, the fistula was excised down to the pancreas. The pancreatic duct was cannulated with a "vitallium tube," and a pancreaticogastrostomy was done. Slight drainage occurred, but the wound was completely healed by the eighteenth postoperative day, and the patient left the hospital on the twenty-third postoperative day. Correction of this complication brought about remarkable changes in his general condition. His appetite improved, he gained in

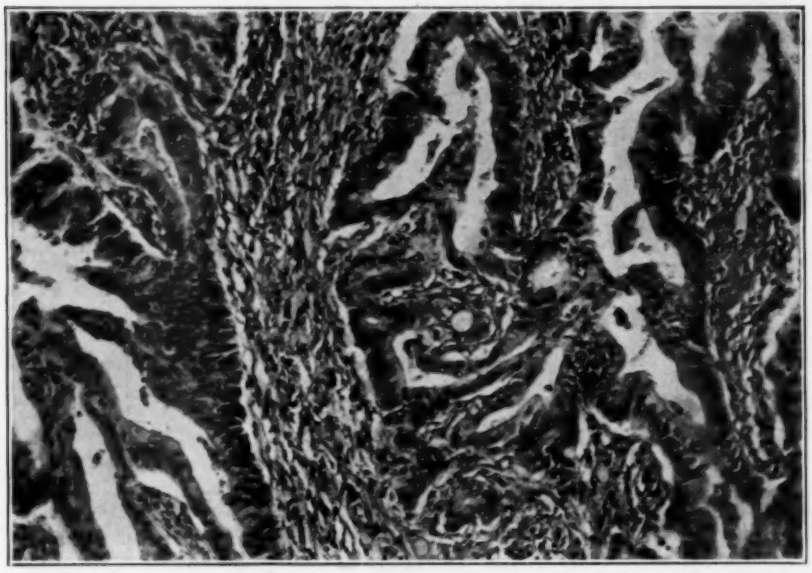

Fig. 3.—Photomicrograph of the adenocarcinoma of the ampulla of Vater.

weight and strength and the blood count and blood plasma proteins returned to normal values.

He was observed periodically in the Surgical Follow-Up Clinic, and a roentgenogram of his abdomen taken on June 30, 1941, showed the "vitallium tube" to be in place; however, a roentgenogram taken on December 15 revealed the disappearance of the tube. It was assumed that it passed into the stomach and was expelled from the body. No untoward reactions were noted from the displacement of the cannula.

For the following two years the patient was asymptomatic. On May 17, 1944, he was admitted to the medical service, with a diagnosis of acute coronary occlusion. The diagnosis was confirmed by electrocardiographic study. He survived this condition and was apparently well except for minor abdominal distress.

On Dec. 12, 1945, he had a rapidly growing papilloma of the skin of the neck removed. On pathologic study this was diagnosed as a questionable melanoma (fig. 4). Attention was not given to this detail.

His progress was satisfactory until his readmission to the hospital on Nov. 28, 1946. He had suffered acute discomfort in the upper abdominal area a week prior to his admission and on the day before had noticed jaundice. At this time the edge

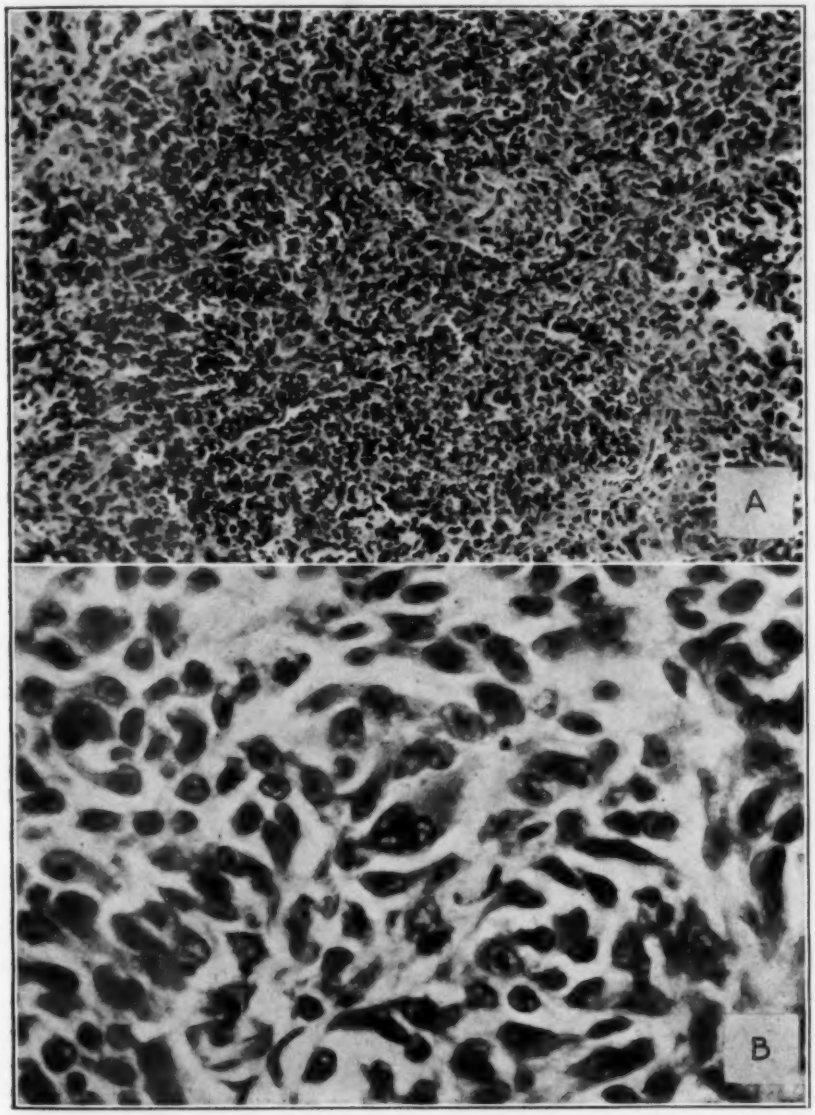

Fig. 4.—Photomicrograph of melanoma of the skin of the neck. *A*, low magnification; *B*, high magnification.

of the liver was 5 cm. below the costal margin, and a questionable firm mass could be felt below the upper portion of the old scar near the right rectus muscle. Laboratory studies indicated normal hepatic function. The icteric index was 32, and

it rose gradually. The stools were clay-colored and contained no blood or urobilin. His blood urea nitrogen content began to rise. We could not explain his obstructive jaundice, but we believed that perhaps he had a stricture at the site of the previous cholecystojejunostomy. On Dec. 11, 1946, an exploratory laparotomy was done under local anesthesia. The gallbladder was extremely firm, and in the region of the junction of the cystic and comon ducts nodular areas could be felt. At the bed of the gallbladder and in the hepatic substance an area could be seen which looked not unlike an area of metastatic carcinoma. A biopsy of material from the liver was done, and the diagnosis of metastatic carcinoma was confirmed by frozen section. The abdomen was closed, and the patient died the following day.

Permission for necropsy was granted, and unusual observations were made. The liver weighed 2,750 Gm., and many matted nodules measuring up to 2.5 cm.

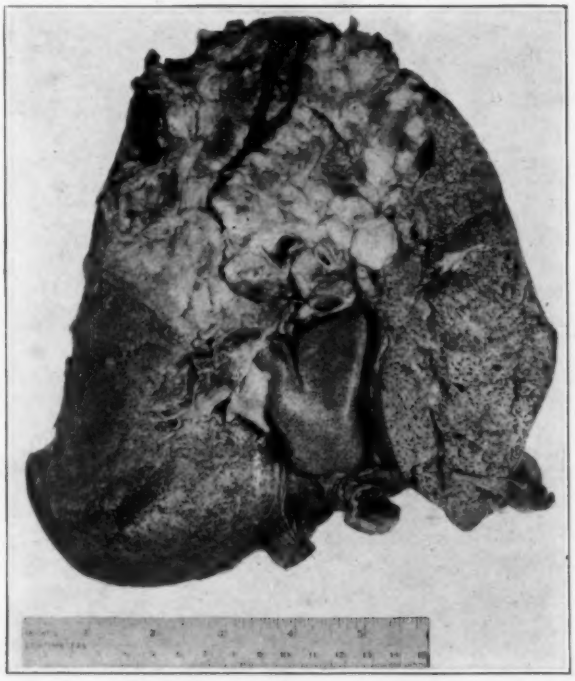

Fig. 5.—Gross specimen of the liver, showing metastatic melanosarcoma infiltrating the porta hepatis.

in diameter were clustered about the porta hepatis. The ducts in the region of the hilus were surrounded by invading neoplasm (fig. 5). The bed of the gallbladder and the gallbladder itself had been replaced by indurated neoplastic tissue. Obstruction of biliary flow due to this growth was seen at the cystic duct. Micropathologic analysis revealed hydrohepatosis and biliary cirrhosis associated with cholangitis. Further study of the tumor confirmed the diagnosis of metastatic melanosarcoma of the liver and its extrahepatic bile duct system (fig. 6). There was no cytopathologic correlation between the primary adenocarcinoma of the ampulla of Vater and the metastatic melanosarcoma of the liver and bile ducts. Carcinoma of the prostate was found in the routine study of this organ (fig. 7).

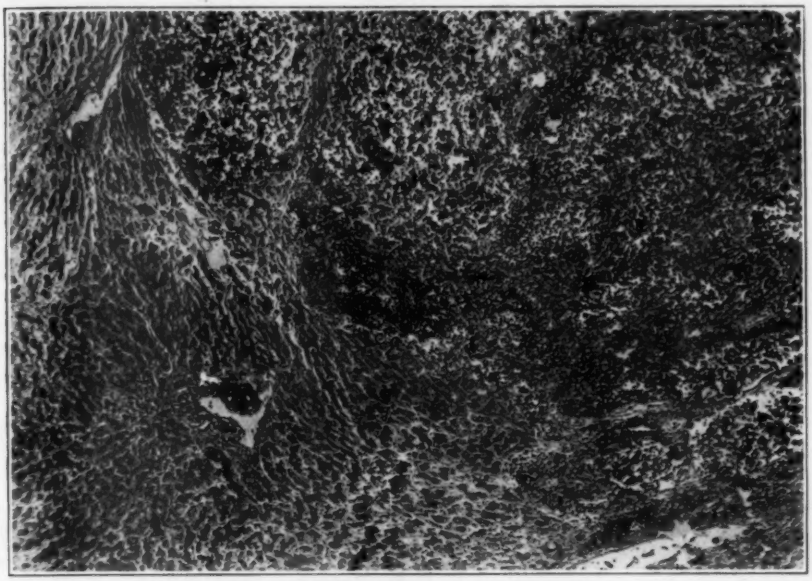

Fig. 6.—Photomicrograph (low power magnification) showing metastatic melanosarcoma infiltrating hepatic substance.

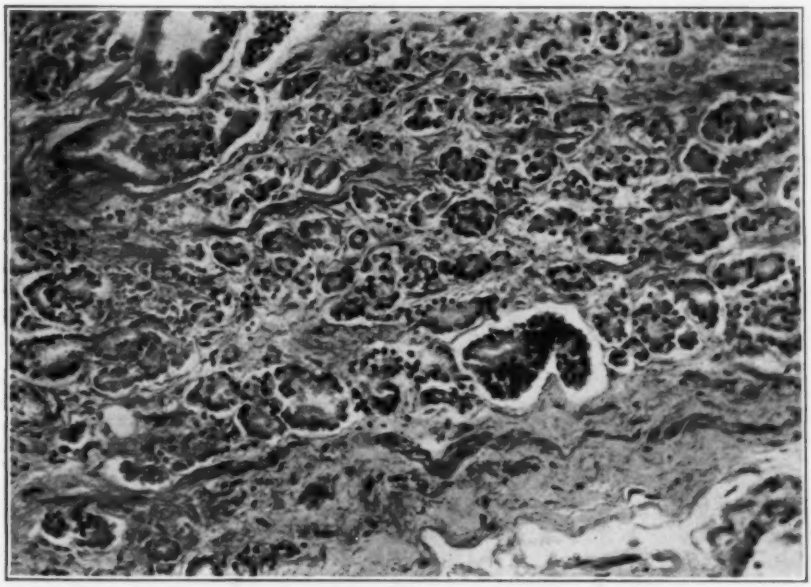

Fig. 7.—Photomicrograph (low power magnification) of carcinoma of the prostate.

This patient survived radical duodenopancreatectomy for seven years and three days. His postoperative course was complicated by a pancreaticocutaneous fistula, which was corrected by a pancreaticogastrostomy. He survived a moderately severe coronary occlusion and died from metastasis of melanosarcoma to the liver and bile duct, the primary site of which was the skin of the neck. A third primary tumor, carcinoma of the prostate, was found at autopsy.

Case 8 (table 3).—A white male waiter was 39 years old at the time of his admission to the Cincinnati General Hospital on March 24, 1944. He complained of constant epigastric pain of two months' duration, accompanied with diarrhea, light-colored stools, weakness, loss of 20 pounds (9 Kg.) in weight and jaundice. At the onset of his illness, three months previously, he suffered fever and chills. There was no nausea or vomiting.

On his admission, he appeared well nourished, and there was no evidence of much loss of weight. The blood pressure was 120 systolic and 70 diastolic. He was deeply jaundiced. The general physical condition was normal except for the abdomen, in which was revealed a smooth, palpable liver extending 6.5 cm. below

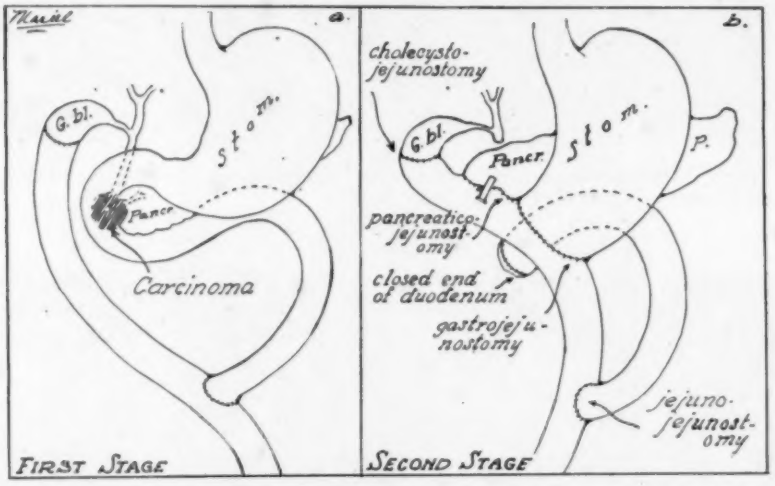

Fig. 8 (case 8, table 3).—*a*, first stage Whipple operation, showing end to end cholecystojejunostomy and end to side jejunojejunostomy; *b*, second stage Whipple operation, showing the partial duodenopancreatectomy (shaded area), end to side gastrojejunostomy and pancreaticojejunostomy.

the costal margin. The gallbladder was not palpable, and no other masses could be felt.

The significant laboratory data were as follows: The blood count was essentially normal. The reaction to the Kahn test was negative and the icteric index was 75. The urine gave a negative reaction for urobilin and a positive reaction for bile. The stool was clay-colored and gave an occasional positive reaction to the guaiac test. The prothrombin time was 95 per cent normal. The blood phosphatase was 12.9 Bodansky units and the reaction to the cephalin flocculation test was negative after forty-eight hours. The blood cholesterol was 727 mg. per hundred cubic centimeters and the serum protein 6.5 Gm. Roentgenographic examination of the stomach and duodenum revealed a "filling defect" in the periampullary region, and a diagnosis of carcinoma of the ampulla of Vater was made.

On April 8, 1944, an exploratory laparotomy was performed and a tumor was palpated in the region of the ampulla of Vater. The clinical impression of pan-

creatitis was confirmed by firmness throughout the pancreatic structure. The first stage of a partial duodenopancreatectomy similar to that described in case 1 was done. An end to end cholecystojejunostomy, in which the Halsted technic was

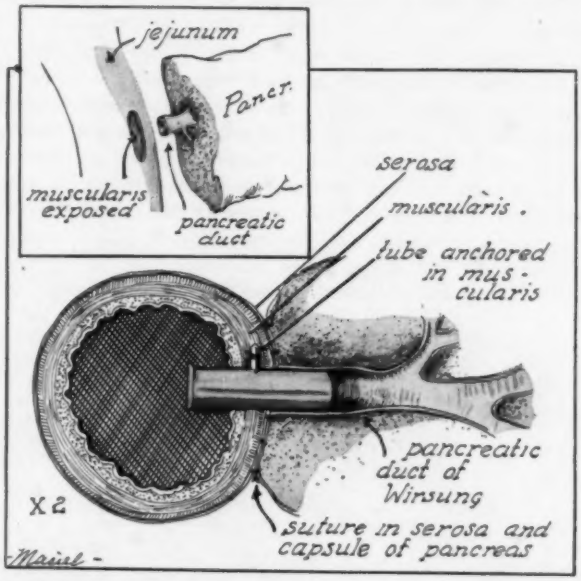

Fig. 9.—Detailed diagram of the pancreaticojejunostomy, showing the implanted "vitallium tube."

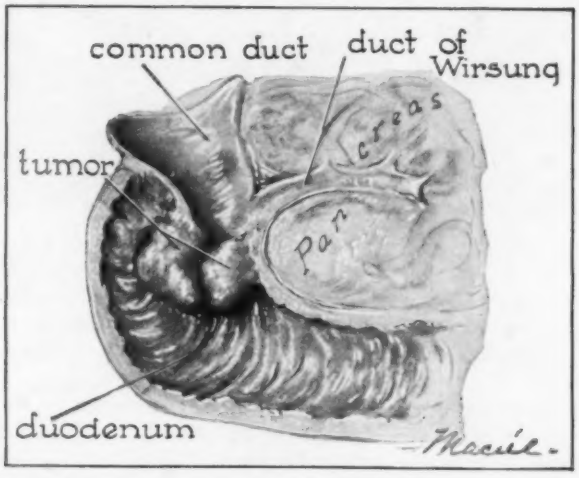

Fig. 10.—Drawing of the gross specimen, showing adenocarcinoma of the ampulla of Vater.

used, and an end to side jejunojejunostomy, with the same approach, were performed. The common duct was not ligated (fig. 8 *a*).

The patient did well postoperatively. The icteric index dropped quickly to 18, and the stools were normal in color. Persistent chronic pancreatitis was shown by increased serum amylase content on repeated determinations.

The patient's condition was excellent, and on April 29 the second stage of the operation was carried out (fig. 8*b*). This consisted in division of the pyloric end of the stomach and inversion of the distal end of the pylorus. The common duct was divided distal to its junction with the cystic duct and ligated with braided silk. A partial duodenopancreatectomy was done after division of the third portion of the duodenum and inversion of the distal end. On resection of the head of the pancreas the duct of Wirsung was easily identified. From the spurt of pancreatic juice an increased intralumenary pressure could be detected. Transection of the duct was performed so that 5 to 6 mm. extended distal to the cut surface of the pancreas. An end to side pancreaticojejunostomy (fig. 9) was done, and a "vitallium tube" 5 mm. in diameter was placed in the pancreatic duct. An end to side gastrojejunostomy was easily accomplished, the left limb of the jejunum and duodenum

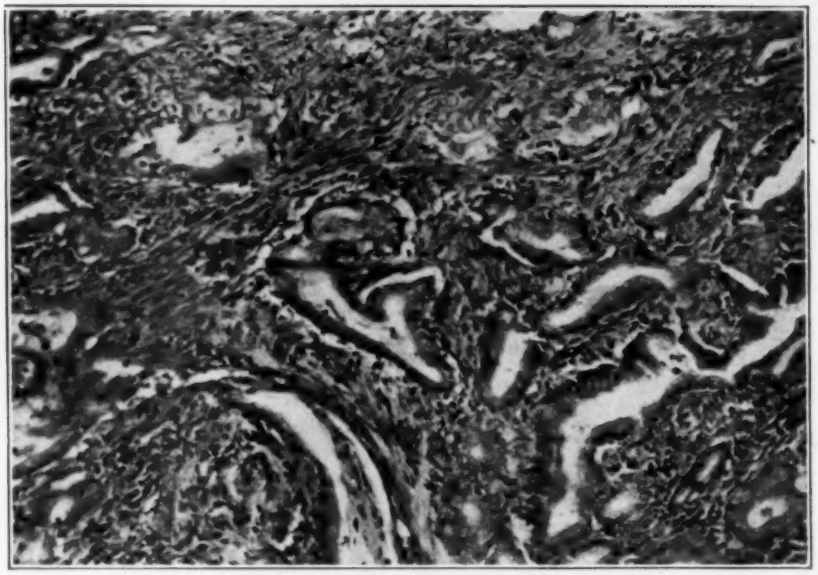

Fig. 11.—Photomicrograph of the adenocarcinoma of the ampulla of Vater.

being retained as a blind, nonfunctioning unit. Drains were placed to the retroperitoneal area of resection and exteriorized through a stab wound. The long transverse incision was closed in layers with silk. The resected tissue is shown in figure 10.

Examination of the specimen showed a papillary adenocarcinoma (fig. 11) of rather low grade malignancy arising from the ampulla of Vater. No metastases were found in any of the lymph nodes removed.

This patient did well postoperatively. The icteric index continued to decline, and the serum amylase content fell to a normal level within four days. The abdominal wound healed by primary union, and he was discharged from the hospital twenty-three days after the second operation.

The "vitallium tube" was seen in place in a roentgenogram taken on June 1, 1944, but was not present in one taken on Sept. 18, 1944.

The patient has been observed closely in the Surgical Follow-Up Clinic, and except for occasional mild epigastric distress he has had no complaints. In November 1946 the results of tests for hepatic function were within normal limits, the icteric index was 5 and a roentgenologic survey revealed the gastrojejunostomy stoma to be functioning well. He is living and apparently well, approximately two years and ten months after operation.

This patient was a younger person in whom adenocarcinoma of the ampulla of Vater occurred and was successfully treated by radical partial duodenopancreatectomy. In this case a pancreaticojejunostomy was done, a procedure which we believe to be desirable.

CARCINOMA OF THE EXTRAHEPATIC BILE DUCTS

A review of the literature clearly demonstrates a lack of experience in the management of carcinoma of the extrahepatic bile ducts, because it has not been widely discussed as a single entity. We have observed and treated 10 patients proved to have carcinoma at this site, and we believe that there are some points of analysis worthy of consideration.

There is a tendency to use the terms "extrahepatic bile ducts" and "common bile duct" synonymously. We believe the former to be anatomically correct. Reference should be made to the specific portions of the duct system when possible, namely, the hepatic ducts (right and left), the common hepatic duct, the cystic duct and the common duct. The last refers to that portion of the duct system starting at the confluence of the common hepatic and the cystic ducts and extending to the ampulla of Vater.

Incidence.—In 1940 Stewart, Lieber and Morgan [11] reported that the incidence of carcinoma of the extrahepatic bile ducts was 0.0135 per cent of all cases of carcinoma studied at necropsy and 0.11 per cent of all cases at the Philadelphia General Hospital. At the same time they found an incidence of 0.028 per cent at the necropsies at Jefferson Hospital, Philadelphia. In a report on 10 cases observed on the Surgical Service, Cincinnati General Hospital, between 1937 and 1946, during which time 31,935 patients were admitted to this service, a clinical incidence of 0.056 per cent was noted for this lesion.

In this group of patients (table 4) the average age was 61.5 years. The oldest patient was 74 years, and only 2 patients were under 60, these being 40 and 57 years respectively. Of these 10 patients, 8 were white and 2 were Negroes; 6 were men and 4 were women, affording a ratio of 3 to 2.

Pathologic Process.—Carcinomas of the extrahepatic bile ducts may be classified as (1) papillary or (2) nodular. The latter type is the one most frequently found. According to Walters and Olson,[12] papillary

11. Stewart, H. L.; Lieber, M. M., and Morgan, D. R.: Carcinoma of Extrahepatic Bile Ducts, Arch. Surg. **41**:662-713, 1940.

12. Walters, W., and Olson, P. F.: Proc. Staff Meet., Mayo Clin. **10**:287-288, 1935.

adenocarcinoma of this region is extremely rare. It has been stated that the tumors arising in the region of the confluence of the three ducts, the hepatic, the cystic and the common, are almost always of the nodular type. There is little question about the existence of a diffuse spreading type of carcinoma in this site. It is known that local extension does occur superiorly and inferiorly. Carcinomas of the extrahepatic bile ducts apparently do not ulcerate as commonly as those in the ampulla of Vater,and bleeding into the gastrointestinal tract is not as frequently observed.

Signs and Symptoms.—The onset of carcinoma of the extrahepatic bile ducts is frequently acute. Irrespective of the mode of onset, the principal symptoms are pain, jaundice, loss of weight, weakness, fever, chills, vomiting and occasionally steatorrhea.

Pain is said to occur in about 53 per cent of the cases. It may be described as a sense of weight or fulness in the epigastrium or as a

TABLE 4.—*Distribution of Age, Sex and Race in Carcinoma of the Extrahepatic Bile Ducts*

Case	Age, Yr.	Race		Sex	
		White	Negro	Male	Female
1	61	+	..	+	..
2	61	+	..	+	..
3	63	+	..	+	..
4	74	+	..	..	+
5	60	+	..	..	+
6	63	..	+	..	+
7	40	..	+	+	..
8	57	+	..	+	..
9	73	+	..	..	+
10	62	+	..	+	..
	61.4	8	2	6	4

constant dull aching type of pain. The history of pain radiating through the back in patients of this age group may be helpful in the early diagnosis of this lesion.

As in carcinoma of the ampulla of Vater, anorexia and loss of weight may be early features of carcinoma of the extrahepatic bile duct. Chills and fever may be observed in the early course of the disease, particularly if there is an associated condition of infection of the gallbladder or of stones in the common duct.

Jaundice usually occurs in the course of this disease and may be seen early and late. Watson [13] stated that complete obstruction of the flow of the bile exists clinically in over 90 per cent of the cases.

The presence of a distended gallbladder will depend on the location of the obstructing carcinoma. Since many lesions develop at the con-

13. Watson, C. J.: Regurgitation Jaundice: Clinical Differentiation of Common Forms, with Particular Reference to Degree of Biliary Obstruction, J. A. M. A. **114**:2427-2432 (June 22) 1940.

fluence of the cystic, hepatic and common ducts, its presence is not as frequent as in carcinoma of the ampulla of Vater. Courvoisier's sign is present in about 26 per cent of the patients.

Laboratory Features.—The laboratory tests done on patients with this condition are identical to those used in ampullary carcinoma. There are, however, one or two striking differences to be noted. Daily examinations of the stool should be made for occult blood; however, the absence of blood in the stool when there are other signs of carcinoma in this region does not exclude the diagnosis of neoplasm. The roentgenographic studies in carcinoma of the extrahepatic bile ducts are not as conclusive for diagnosis as in carcinoma of the ampulla of Vater. The absence of "filling defects" and of a "reversed 3 sign" suggests the possibility of carcinoma of the ducts. We have noted an occasional tendency for the superior border of the second portion of the duodenum to be "pulled up" and "flattened," a finding compatible with a growing neoplasm of the duct system.

Differential Diagnosis.—The problem of differential diagnosis is the same as for ampullary lesions, but it should be kept in mind that an accurate clinical diagnosis is not important in the management of the condition. The goal to be attained is early diagnosis of suspected carcinoma in this region, followed by early exploration. Only when this principle is followed will the resectability rate improve.

Treatment.—Radical treatment of carcinoma of the extrahepatic bile ducts cannot be done as frequently as desired. When the lesion occurs in any portion of the common duct, resection may be done, but in ascending the duct system one finds wide excision increasingly more difficult, until finally it is technically impossible. Partial choledochoduodenopancreatectomy is the most desirable operation.

Analysis of Cases and Results (*table 5*).—Ten cases of proved carcinoma of the extrahepatic bile ducts are reported. Of these, in only 1 (case 8) was a partial choledochoduodenopancreatectomy possible. In addition, a palliative operation was feasible in only 1 case (case 3). Actually, the first stage of a Whipple procedure was done, but the carcinoma spread locally, preventing further treatment. Six of the patients submitted to exploratory laparotomy and to biopsy of the lesion. Either the extent of the local lesion or metastases to the liver and peritoneum precluded even palliative intervention. One patient (case 6) had her illness complicated by a manic-depressive psychosis. and operation was delayed pending authorization by relatives. Another patient whose condition was never favorable for operation submitted to peritoneoscopy and died during the procedure.

Analysis of the survival period indicates carcinoma of the extrahepatic bile ducts to be extremely malignant in its course. When

TABLE 5.—*Carcinoma of Extrahepatic Bile Ducts*

Case	Age, Yr.	Race	Sex	Date of Admission	Operative Procedure *		Exploratory Laparotomy	Result		Period of Survival			Comment
					Duodeno-pancreatectomy	Palliative		Living	Dead	Yr.	Mo.	Days	
1	61	White	Male	3/31/39	.....	.....	Biopsy only, 4/11/39	...	5/21/39	..	..	40	Bile duct carcinoma—proved by biopsy; mass greatest at junction of cystic and common ducts; no autopsy
2	61	White	Male	4/ 9/40	.....	.....	Biopsy only, 5/25/40	...	5/29/40	..	..	4	Bile duct carcinoma—proved by autopsy; single metastasis to liver
3	63	White	Male	12/27/40	.....	First stage Whipple 1/14/41	Obstruction due to neoplasm at cystic and common ducts, 2/11/41	...	3/ 8/41	..	..	53	Bile duct carcinoma—proved by autopsy; tumor spread by direct invasion
4	74	White	Female	9/12/41	.....	.....	Biopsy only; stone had ulcerated through gooseneck of gallbladder, 9/12/41	...	12/13/41	..	..	92	Bile duct carcinoma—proved by biopsy; stone in gallbladder had caused gooseneck perforation; no autopsy
5	60	White	Female	11/ 5/41	.....	.....	Biopsy only; obstruction at junction of cystic and common ducts; carcinoma invading gallbladder, 11/17/41	...	12/31/41	..	..	44	Bile duct carcinoma—proved by biopsy; common duct, liver and gallbladder involved; no autopsy
6	63	Negro	Female	12/ 8/41	.....	.....	No operation; manic-depressive psychosis	...	2/ 9/42	..	..	..	Bile duct carcinoma—proved by autopsy; carcinoma in hepatic duct; patient's condition never permitted exploration
7	40	Negro	Male	6/27/42	.....	.....	Widespread metastases to liver, 8/15/42	...	8/20/42	..	..	5	Bile duct carcinoma—proved by autopsy; liver revealed widespread metastases
8	57	White	Male	3/24/44	Two stage Whipple † (a) 4/ 4/44 (b) 4/22/44	.....	.....	...	9/18/45	1	4	26	Bile duct carcinoma—proved by resected tissue; patient lived over 1 yr. and 4 mo.; no autopsy
9	73	White	Female	5/ 8/45	.....	.....	Peritoneoscopy—patient died during this procedure, 5/25/45	...	5/25/45	..	..	..	Bile duct carcinoma—proved by autopsy; involvement of hepatic duct with direct extension into liver
10	62	White	Male	7/ 1/46	.....	.....	Biopsy only, 7/24/46	...	8/13/46	..	..	19	Bile duct carcinoma—proved by biopsy; metastases to liver and early carcinomatosis; no autopsy
		10			1	1	7	0	10				

* The operability rate was 10 per cent for choledochoduodenopancreatectomy, 10 per cent for palliative procedures and 80 per cent for exploratory laparotomy. The mortality rate (ten year period) was 100 per cent.

† Described in the case report.

abdominal exploration alone was done the average survival period was thirty-four days. One patient (case 8) in whom a partial duodenopancreatectomy was performed survived for one year and five months.

In this group of 10 cases the operability rate was as follows: for radical resection, 10 per cent; for palliative operations, 10 per cent, and for exploratory laparotomy, 80 per cent. The mortality rate during this ten year period was 100 per cent.

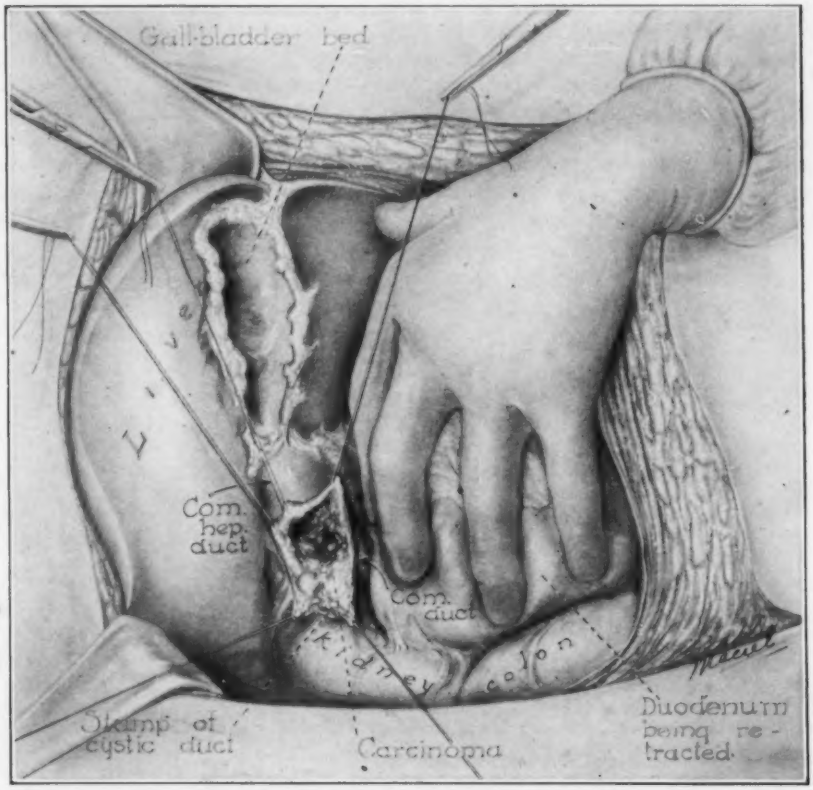

Fig. 12.—Drawing of liver and common duct at the time of operation, showing the location of the carcinoma.

We believe that several factors account for the lack of correct diagnosis in carcinoma of the extrahepatic bile ducts. In this connection, the following case report is of interest.

Case 8 (table 5).—A white man suffering from a manic-depressive psychosis was 57 years old at the time of his admission to the Cincinnati General Hospital on March 24, 1944. He complained of gradual loss of weight (35 pounds [16 Kg.] in one year), followed by anorexia and jaundice associated with a constant boring pain in the upper lumbar region of the spine. There was no nausea, vomiting or steatorrhea. On his admission to the hospital he appeared moderately nourished, with evidence of loss of weight, and profoundly jaundiced.

* The operability rate was 10 per cent for choledochoduodenopancreatectomy, 10 per cent for palliative procedures and 80 per cent for exploratory laparotomy. The mortality rate (ten year period) was 100 per cent.
† Described in the case report.

The blood pressure was 150 systolic and 80 diastolic. An indefinite mass about 3 cm. in diameter was palpated below the right costal margin. The significant laboratory data were as follows: The blood count was essentially normal, and the reaction to the Kahn test was negative. The icteric index was 150. The urine gave a negative reaction for urobilin and a positive reaction for bile. The stool was clay-colored and gave a positive reaction to the guaiac test. The prothrombin time was 75 per cent normal, and the blood plasma protein content was 7.0 Gm. per hundred cubic centimeters. The reaction to the cephalin flocculation test was 3 plus in forty-eight hours. A diagnosis of obstructive jaundice due to neoplasm was made.

On April 4, 1944, an exploratory laparotomy was performed, and a large, tensely distended gallbladder was immediately encountered. On aspiration 135 cc. of thick grayish material was obtained, and a large stone could be palpated in the gooseneck. The head of the pancreas and duodenum seemed normal; however, the region of the cystic and common ducts felt thickened and nodular. Since no bile was obtained, a cholecystectomy was done. When the cystic duct was divided, definite cartilaginous appearance of the tissue was noted. Frozen section of this tissue revealed the presence of carcinoma. A drawing of the liver and of the common duct at the time of the operation is shown in figure 12.

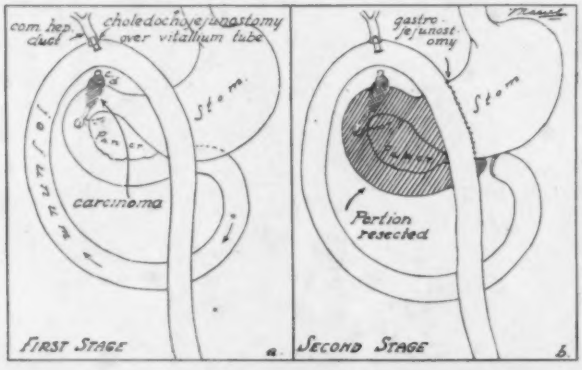

Fig. 13 (case 8, table 5).—*a*, first stage Whipple operation, showing division of the common hepatic bile duct proximal to the tumor and end to side choledochojejunostomy; *b*, second stage Whipple operation, showing partial choledochoduodenopancreatectomy (shaded area) with end to side gastrojejunostomy.

There was no evidence of involvement of local lymph nodes, and no metastasis was noted in the liver. The common hepatic duct was divided in its midportion about 1.5 cm. above the superior border of the neoplasm. An end to side isoperistaltic choledochojejunostomy was done, a "vitallium tube" 6 mm. in diameter being used as a cannula (fig. 13*a*). The distal end of the duct was ligated with heavy braided silk. The patient did well postoperatively, and within three weeks the icteric index dropped to 18. His general condition improved. On April 22 the second stage of the partial choledochoduodenopancreatectomy was done (fig. 13*b*). This consisted of division of the antrum of the stomach, followed by resection of the distal end of the common bile duct (which contained the tumor), the duodenum and the head of the pancreas. This block of tissue was removed intact (fig. 14). The distal end of the fourth portion of the duodenum was closed. The pancreas was resected by dividing tissue between clamps and transfixing these points with medium silk. An end to side gastrojejunostomy was done, with no attempt to implant the duct of Wirsung into the gastrointestinal canal. The

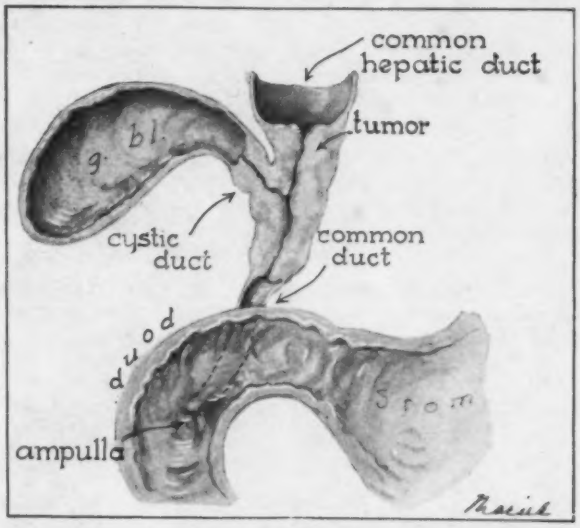

Fig. 14.—Diagrammatic drawing of the sagittal section of the resected tissue, showing the location of the carcinoma involving the confluence of the common duct, common hepatic duct and cystic duct.

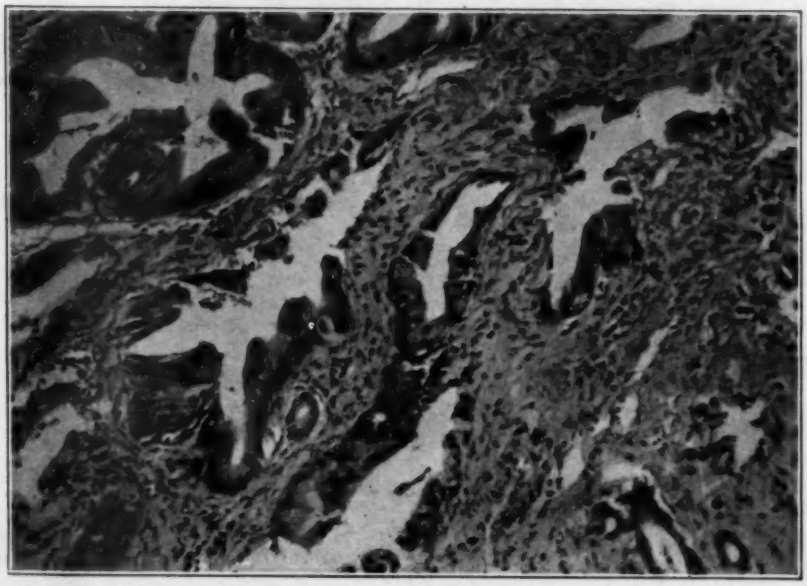

Fig. 15.—Photomicrograph of the adenocarcinoma of the extrahepatic bile ducts.

retroperitoneal area was drained through the working incision and the abdomen closed.

Examination of the specimen showed a carcinoma of the bile duct somewhat anaplastic in its cytologic structure (fig. 15). No metastases to the regional lymph nodes were noted.

This patient did well postoperatively except for discomfort in the midline directly at the level of the resection. The wound healed by primary union. No bile or pancreatic secretion drained; however, the serum amylase content was increased. He was discharged from the hospital two months after resection, with only a mild complaint of epigastric discomfort.

He was observed in the Surgical Follow-Up Clinic, and because of vague abdominal distress repeated roentgenologic studies were made. These revealed the gastrojejunostomy stoma to be functioning well. The "vitallium tube" used as a cannula in the initial choledochojejunostomy was expelled from the body. A roentgenogram (fig. 16*A*) taken on May 15, 1944, revealed its position, whereas

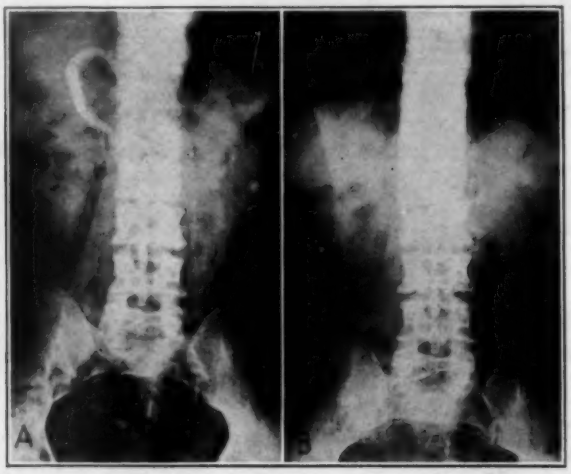

Fig. 16.—*A*, roentgenogram showing the position of the "vitallium tube" used as a cannula in the choledochojejunostomy; *B*, roentgenogram of the abdomen, confirming absence of the "vitallium tube."

it could not be seen in one taken on Aug. 30, 1945 (fig. 16*B*). The patient was returned to the Longview State Mental Hospital, where he died on Sept. 18, 1945. No autopsy was made.

This patient was the only one of 10 patients with carcinoma of the extrahepatic bile ducts in whom radical resection by partial choledochoduodenopancreatectomy was performed. His survival period was one year, four months and twenty-six days.

COMMENT

It is realized that eighteen patients comprise too small a series from which to draw definite conclusions. In addition, the classification of the disease under ampullary carcinoma and carcinoma of the extrahepatic bile ducts is largely of academic interest because the procedure indicated for radical resection varies only in the technical approach. Never-

theless, analysis of our cases classified under these two divisions shows that ampullary carcinoma has a higher rate of resectability than carcinoma arising in the extrahepatic bile ducts. This may be explained in part by an earlier diagnosis and treatment of the former in conjunction with a difference in behavior of growth on the part of the tumor. From a technical viewpoint the latter condition is decidedly more difficult to manage. Both lesions warrant early diagnosis and early exploration. Today the latter can be accomplished with relative safety. The operation of choice is partial choledochoduodenopancreatectomy.

With the two series of cases combined, partial choledochoduodenopancreatectomy was possible in 5 of the 18 cases reported, giving a resectability rate of 27.7 per cent. The comprehensive ten year (1937 to 1946) mortality rate was 94.4 per cent.

SUMMARY AND CONCLUSIONS

1. The incidence, pathologic process, signs and symptoms, laboratory features, diagnosis and treatment of carcinomas occurring in the ampulla of Vater and in the extrahepatic bile ducts have been discussed.

2. Eight proved cases of ampullary carcinoma (table 3) have been reported (2 in detail), with a resectability rate of 50 per cent and a ten year mortality rate of 87.5 per cent. The patient in case 1 lived for seven years and three days, and the patient in case 8 is alive at the time of this report, two years and ten months later.

3. Ten proved cases of carcinoma of the extrahepatic bile ducts (table 5) have been reported (1 in detail), with a resectability rate of only 10 per cent and a ten year mortality rate of 100 per cent.

4. For the combined series (18 cases) there was a resectability rate of 27.7 per cent and a ten year mortality rate of 94.4 per cent.

5. The low resectability rate and the high mortality rate reveal the seriousness of the lesions. The goal must be earlier diagnosis and earlier surgical treatment. Jaundice per se is no longer a contraindication to exploratory laparotomy when proper preoperative therapy has been given.

6. Three cases have been reported in detail. Methods of performing pancreaticogastrostomy and pancreaticojejunostomy have been discussed. A "vitallium tube" used as a cannula implanted in the 3 cases was in each instance expelled from the body.

7. Pancreaticojejunostomy is a desirable feature of partial duodenopancreatectomy because the pancreatic secretion aids digestive processes and its flow to the gastrointestinal tract eliminates the possibility of hydropancreatosis.

8. Consideration of the survival period indicates partial choledochoduodenopancreatectomy to be the procedure of choice.

BILATERAL CERVICAL RIBS ASSOCIATED WITH NEUROPATHIC JOINTS

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IN 1868 Charcot [1] described 4 cases of tabes dorsalis in which, without obvious trauma, joints of the lower extremities became involved in firm, painless swelling, the surfaces undergoing changes which produced false positions and luxations of the bones. Similar changes were described in syringomyelia by Schultze and Kahler,[2] and according to Schlesinger [3] the condition may develop in 30 to 40 per cent of patients with this lesion of the spinal cord. Whereas in tabes the lower extremities are more often affected, in syringomyelia the upper extremities are favored. Such arthropathies following injury of the peripheral nerves were first reported by Packard,[4] who described a case in which a sciatic nerve was compressed by tumor. Other inciting causes which have been found are injury of the brachial plexus and spina bifida.

PATHOGENESIS

The disease usually begins in the cartilage and the zone of calcification between the cartilage and the underlying cancellous bone. Trophic disturbances, secondary to irritative involvement of the sympathetic nerves, plus the effects of minimal trauma lead to serous effusion, laxity of ligaments, erosion of cartilaginous surfaces, sclerosis of subchondral bone, flattening and condensation of bone heads, parosteal bone formation, marginal fractures, loose bodies in the joint, exostoses and calcification of muscular and ligamentous structures.

Read at the fourth annual meeting of the Central Surgical Association, Chicago, Feb. 22, 1947.

1. Charcot, J. M.: Arch. de physiol. norm. et path. **1**:161, 1868.

2. Schultze, F., and Kahler, O., cited by Leede, C. S.: Arthropathien bei Syringomyelie, Munich, Kastner & Callwey, 1908.

3. Schlesinger, H.: Die Syringomyelie, ed. 2, Leipzig, F. Deuticke, 1902, pp. 93-144.

4. Packard, cited by Mitchell, S. W.: Tr. Path. Soc. Philadelphia, 1863.

ROLE OF THE SYMPATHETIC NERVES

Telford [5] believes that only when there is some abnormality in the distribution of sympathetic fibers will the symptoms develop in patients with cervical ribs. He has shown that in the normal persons the sympathetic fibers leading to the blood vessels join the roots of the brachial plexus and are thoroughly mixed with the myelinated nerve fibers by the time these trunks cross the cervical rib. In approximately 1 in 8 persons the sympathetic nerve fibers leading to the blood vessels form a special bundle situated just below the brachial plexus. This bundle of nonmyelinated nerve fibers is in a position to be irritated by a cervical rib since it crosses the rib as an entity before its fibers mingle with those of the larger nerve trunks. It is believed that ribs 4 cm. or more in length are in a position to cause such irritation.

CLASSIFICATION

Shands [6] has given the following etiologic classification of neuropathic joints, which is the most satisfactory coming to our attention:

(1) Tabes dorsalis.

(2) Syringomyelia.

(3) Lesions of the peripheral nerves, which include (*a*) injury, (*b*) peripheral neuritis and (*c*) leprosy.

(4) Lesions of the spinal cord, which include (*a*) injury, (*b*) congenital malformations, (*c*) tumors, (*d*) tuberculosis of the spine, (*e*) acute myelitis, (*f*) anterior poliomyelitis and (*g*) progressive (central) muscular atrophy.

(5) Lesions of the cerebrum, which include (*a*) dementia paralytica and (*b*) hemiplegia following cerebral hemorrhage.

It would appear that the lesion in the case here reported is of type 3 and that the case is similar in several respects to that described by Mitchell,[7] in which, after dislocation of the shoulder and injury of the brachial plexus, extensive changes developed in the joints.

REPORT OF A CASE

A 38 year old white railroad supervisor entered the hospital on Oct. 21, 1946, complaining of pain in the left fourth finger and the fifth fingers.

Past History.—His general health had been excellent. He had never had any serious illnesses and denied the venereal diseases by name.

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5. Telford, E. D., and Stopford, J. S. B.: Brit. J. Surg. **18**:557, 1931.

6. Shands, A. R.: Neuropathies of the Bones and Joints: Report of Case of Arthropathy of Ankle Due to Peripheral Nerve Lesion, Arch. Surg. **20**:614 (April) 1930.

7. Mitchell, S. W.: Injuries of Nerves and Their Consequences, Philadelphia, J. B. Lippincott Company, 1872.

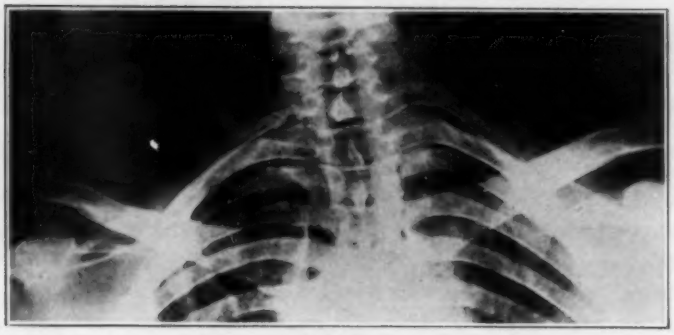

Fig. 1.—Roentgenogram showing bilateral cervical ribs.

Fig. 2.—*A*, roentgenogram of right elbow joint; *B*, roentgenogram of left elbow joint.

Present Illness.—In December 1942, while driving his car with both hands exposed on the steering wheel, he first observed atrophy of the interosseous muscles between the thumb and the index finger of the left hand. During that winter the fourth and fifth fingers of the left hand would flex involuntarily during exposure to cold, later relaxing when warmed. He next experienced tingling sensations over the left fifth finger and over the ulnar half of the fourth finger. While playing golf he noticed that the handle of his club would rotate involuntarily in his left hand. In May 1943 he visited a southern clinic, where section of the left scalenus anticus muscle was done. No roentgenogram of the neck or shoulder was taken. He had immediate relief from the tingling sensations, but the strength of the muscles did not return and during the following year atrophy progressed, with involvement of all the interosseous spaces of the left hand. During the summer of 1946 shooting pains developed over the posterior surface of the left shoulder. These pains were not influenced by the weather.

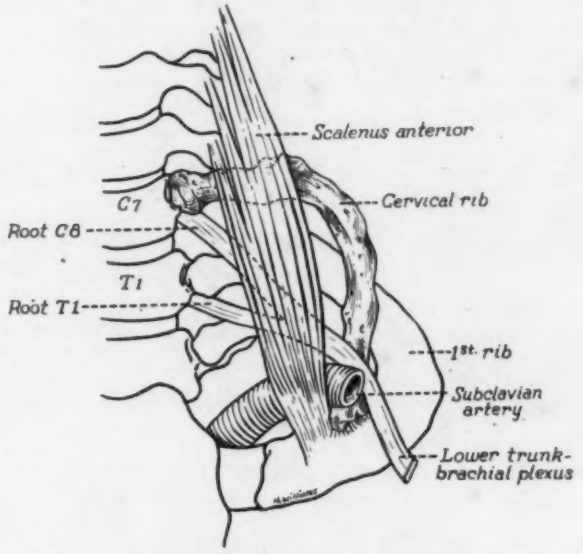

Fig. 3.—Tough ligamentous attachment of cervical rib to first rib.

Physical Examination.—The patient was a handsome athletic type of man, impressing one immediately with his vitality and muscular development. Palpation in the supraclavicular fossae revealed unmistakable bilateral cervical ribs, which were nearly visible. Pressure over the left rib elicited shooting pain over the posterior surface of the left shoulder. There was considerable atrophy of the left spinati and serratus anterior muscles, with lateral displacement of the scapula and slight winging of the angle. The extensor muscles of the left forearm were weak. Interosseous atrophy was present to a notable degree in the entire left hand. The blood pressure was 132 systolic and 84 diastolic in both arms. There were no changes in color or sensation. Both elbows lacked the last 15 degrees of normal flexion, but they were not swollen or otherwise abnormal. Lateral mobility was slightly increased. The joints were aspirated with a no. 19 needle, and it was noted that there was complete absence of pain as soon as the skin had been penetrated.

Laboratory Observations.—The hemoglobin content was 15.2 Gm. per hundred cubic centimeters, the red blood cell count 5,360,000 and the white blood cell count 7,000, with a normal differential count. Urinalysis and Wassermann and Kline tests of the blood revealed nothing abnormal.

Roentgenologic Findings.—Roentgenograms confirmed the finding of bilateral cervical ribs, the left being estimated at 4 inches (10 cm.) in length and the right at 5 inches (12.7 cm.). Both elbow joints showed extensive roughening and thickening of their articular surfaces, with the presence of osteophytes and loose bodies of rounded contour.

Operation.—On Oct. 22, 1946, with the patient under cyclopropane and ether anesthesia, the left cervical rib was resected through an anterolateral approach. The anterior end of the rib was rigidly bound to the first rib by a tough ligamentous band, and it was evident that section of the anterior scalenus muscle could not have altered the position of the first rib.

Subsequent Course.—On the day after operation the patient spontaneously mentioned the absence of pain in the posterior part of the shoulder, and a week later he reported complete freedom from pain in the fourth and fifth fingers. When he was seen four months later sensation was perfectly normal and muscular strength had improved greatly.

COMMENT

In addition to demonstrating the occurrence of bilateral neuropathic elbow joints associated with bilateral cervical ribs, the case discussed also shows the failure of scalenotomy alone to give relief to patients with cervical ribs which have dense though radiologically invisible attachments to the first rib. Such cases emphasize the importance of a wide exposure and thorough operative investigation of the relationships before simple section of the muscles alone is performed. In a number of cases resection of the ribs is required rather than blind scalenotomy through a small incision.

FEMORAL HERNIA

Treatment and Results

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WHEN an irreducible hernia is present in the groin, the chances are even that it is a femoral hernia. Femoral hernias, despite their rarity, commonly represent surgical emergencies, whereas strangulation occurs in only 1 per cent of inguinal hernias. It is not unusual for a femoral hernia to make its first appearance in the form of strangulation. In fact, this occurred in about 50 per cent of the cases presented. Because of this disposition to early incarceration and strangulation, the mortality of femoral hernia has been relatively high.

In a study of the relative frequency of femoral and inguinal hernia the incidence was found to vary from 2 per cent of all hernias at the Henry Ford Hospital [1] to 5 per cent at St. Luke's Hospital, New York [2]; studies at both hospitals covered the same twenty year period. The combination of a low comparative incidence with a high incidence of strangulation and higher mortality rates has stimulated a study of cases of femoral hernia. The relatively high recurrence rate after operation has prompted the application of newer technics in an effort to effect a higher percentage of successful repairs. Burrows [3] stated several years ago that there were over one hundred advocated operations for femoral hernia, and since that time several more have been reported. This supports the assertion made by others that currently used operations are not giving entirely satisfactory results. That this statement bears some truth is indicated by the few follow-up studies published. Among the many series reported, in less than one in four are any statistics given in relation to recurrence.

In the past six years I have treated one hundred and forty-nine hernias surgically, and of these, fifteen, or 10 per cent, have been

Read at the fourth annual meeting of the Central Surgical Association, Chicago, Feb. 22, 1947.

1. McClure, R. D., and Fallis, L. S.: Femoral Hernia, Ann. Surg. **109**:987-1000, 1939.

2. Shelley, H. J.: Femoral Hernias: Study of Two Hundred and Thirty-Eight Cases and Two Hundred and Twenty-Six Repairs, Arch. Surg. **41**:1229-1243 (Nov.) 1940.

3. Burrows, S. J.: Femoral Herniorrhaphy, Am. J. Surg. **40**:451-453, 1938.

femoral hernias. Over a nine year period there have been 19 consecutive cases in which femoral hernia has been subjected to operation. These form the basis of this report.

OCCURRENCE

The average age of 19 patients was 62 years; 3 were 80, and the youngest was 30. The hernias occur in middle-aged and old patients and are seldom encountered in infancy. Women are more commonly afflicted, and many reasons for this have been advanced. Pregnancy particularly is considered a predisposing factor. The right side is more frequently affected than the left, the hernias occurring on the right side in fully two thirds of the cases. No successful explanation has been given for this any more than for the fact that inguinal hernia favors the right side. According to Ritchie,[4] one third of all hernias in women and 6 per cent of all hernias in men are femoral.

CAUSATION

The question of causation is admitted to be controversial, and several factors are worthy of consideration. Since congenital or preformed diverticula have been found at postmortem examinations in the femoral canal, the presence of this is the cause most generally accepted. The majority of these hernias arrive in the later decades of life, when tissues are more atrophic and less elastic and when increases in pressure in the lower part of the abdomen from other causes are common. This factor, along with a loss of fat about the femoral canal and an increase in the size of the normal femoral fossa, may be the cause of many of the hernias.

The association of inguinal and femoral hernias has been mentioned by McClure and others. The same factors probably operate to produce the two types, namely, a congenital sac or an increase in abdominal pressure or both. Femoral hernia has been observed to appear after operation for inguinal hernia and vice versa. Watson[5] stated that in the usual method of repair of inguinal hernia, Poupart's ligament is pulled upward, the size of the femoral ring thus being increased. I have observed the appearance of a femoral hernia on the same side three years after repair of inguinal hernia but have also observed the appearance of bilateral femoral hernias in the presence of untreated inguinal hernia.

ANATOMIC CONSTRUCTION OF THE AREA

In more recently advocated operations the anatomic construction about the femoral ring has received increased attention. The ring is

4. Ritchie, W. P.: Essentials of General Surgery, St. Louis, C. V. Mosby Company, 1941.

5. Watson, L. F.: Hernia, St. Louis, C. V. Mosby Company, 1924.

formed by Poupart's or the inguinal ligament anteriorly, the pectineal fascia, the origin of the pectineus muscle and Cooper's ligament posteriorly, the femoral vein laterally and the lacunar or Gimbernat's ligament below and medially. The femoral canal is normally a small and relatively unimportant space, difficult in ordinary circumstances to demonstrate unless occupied by a hernia or tumor. The superior pubic ligament, or Cooper's ligament, deserves particular description since it has been brought so forcefully into the consideration of hernia repair by the studies of McVay and his associates.[6] In recent literature on hernial repair Cooper's ligament as a corporate anchor for the transversalis fascia has been described as of outstanding importance. This superior pubic ligament forms a remarkably strong fold above the ileopectineal line, extending from the tuberosity of the pubis outward and projecting from the bone over that line.

DIAGNOSIS

Femoral hernia when discovered in routine examination should be noted and surgical repair advised because of the possibility of strangulation. The diagnosis is not always easy, particularly as regards the differentiation from inguinal hernia. This differentiation is important, however. A femoral hernia is lateral to the pubic spine, whereas an inguinal hernia is above or medial to this landmark. An inguinal hernia usually is reduced when the patient lies down. Routine surgical correction of uncomplicated femoral hernia will prevent strangulation, with its attendant high morbidity, mortality and recurrence rate.

STRANGULATION

The cause of strangulation is not agreed on by all investigators. However, Gimbernat's or the lacunar ligament is a relatively sharp and unyielding structure and is commonly believed to be the chief factor in the prevention of spontaneous or manipulative replacement of the hernia. The narrowness of the sac itself may be an important factor.

Pain and vomiting are the two outstanding and common symptoms ushering in strangulation. They may be sudden or gradual in onset and may even be chronic, persisting for many days or weeks. Usually the pain is generalized over the abdomen as in intestinal obstruction. It varies considerably in intensity, and in general the dictum "the more severe the symptoms the more severe the disease" holds true. When circulatory disturbance of the strangulated tissue occurs, more severe signs and symptoms may be expected. Of 12 patients with strangulated hernias in this series, 9 complained particularly of severe abdominal col-

6. McVay, C. B., and Anson, B. J.: Fundamental Error in Current Methods of Inguinal Herniorrhaphy, Surg., Gynec. & Obst. **74**:746-750 (March) 1942.

icky pains, with vomiting and pain which was referred to the hernia. One presented symptoms of acute intestinal obstruction without symptoms referred to the hernia. The majority of strangulated hernias may be diagnosed without undue difficulty from the history and the abdominal and crural examination. There is one type of strangulated femoral hernia which does not present this ease of diagnosis. It is frequently overlooked or treated under another working diagnosis. It is worthy of special study and generally possesses the following characteristics:

A. It is apt to occur in persons from 70 to 80 years of age.

B. The symptoms are slow in onset, frequently cannot be dated exactly and in general consist of mild intermittent colicky abdominal pains, intermittent vomiting, constipation, loss of appetite, loss of weight and weakness.

C. On examination the evidence is not alarming. The abdomen is soft and may be slightly distended. Usually if the hernial rings are carefully examined a small femoral hernia may be demonstrated. It will not be painful, and on being questioned the patient may state that the lump has been there for years and has never caused any trouble. The hernia, if noticed, may appear so innocent that it may be looked on as merely a coexistent lesion. Often the hernial rings are not examined, and the hernia is not considered in the diagnosis. Other conditions, such as carcinoma of the large bowel, may be suspected as the cause of the low grade intestinal obstruction. Dehydration appears within a short time, and the patient's illness takes a gradual but steady downhill course. Because these patients are fairly old, irreversible changes in blood and in cellular metabolism take place, with resultant protein deficiency, and they become poor operative risks when the correct diagnosis is made.

Douglas,[7] McNealy[8] and Dunphy[9] have emphasized this type of hernia in their writings, and the last author stated that for 29 of 51 patients with strangulated femoral hernias entering Peter Bent Brigham Hospital an incorrect diagnosis had been made before admission. These patients less often have loss of viability of the strangulated tissue, and consequently the rapid pulse and other signs of shock do not occur. The mortality is in direct proportion to the number of days which have elapsed since the beginning of obstruction and to the presence or loss of tissue viability of the strangulated contents. Statistics emphasize

7. Douglas, D. M.: Femoral Hernia with Factors Affecting Mortality, Brit. M. J. **1**:354-357, 1942.

8. McNealy, R. W.; Lichenstein, M. E., and Todd, M. A.: Diagnosis and Management of Incarcerated and Strangulated Hernia, Surg., Gynec. & Obst. **74**:1005-1010, 1942.

9. Dunphy, J. E.: Diagnosis and Surgical Management of Strangulated Femoral Hernia, J. A. M. A. **114**:394-396 (Feb. 3) 1940.

the need of operating early when acute obstruction or circulatory impairment of strangulated contents is prognosticated by the usual signs. Delay will only court disaster.

METHODS OF REPAIR

Mention has previously been made of the number and variety of operative methods advocated for the surgical repair of femoral hernias. The simplest is that of ligation of the hernial sac. In 1906 Ochsner [10] reported 30 consecutive cases in which operation was performed by this method, without recurrence. He advocated it for all the small and average-sized hernias. Burrows [3] and Waugh [11] have in recent articles championed this method, stating that it has given satisfactory results in their hands. Bassini,[12] in a report in 1893 of fifty-four operations without recurrence, impressed the surgical world so greatly that his method has been the most widely used since that time. He excised the sac after ligation and closed the femoral canal by suturing the pectineal fascia to the posterior margin of Poupart's ligament. The falciform process of the fascia lata was sutured to the pectineal fascia. All suturing was done with interrupted sutures.

Other surgeons have approached the problem with a more direct attack on the femoral ring through the inguinal canal. Lotheissen [13] in 1898 described the repair of femoral hernia through the floor of the inguinal canal, the conjoined tendon being sutured to Cooper's ligament to produce a barrier before the femoral ring. The same result can be obtained by suturing the mobilized external oblique tendon or, more preferably, the transversalis fascia to Cooper's ligament to produce this barrier. The methods of inguinal approach, with minor variations in technic, have the same aims: (1) to obtain a clearer view of the femoral ring and the contents of the femoral sac; (2) to correct and remove inguinal hernias if present (McClure advises routine digital examination intraperitoneally of the femoral ring through the opened indirect hernial sac in operations for inguinal hernia, which is known as Hoguet's maneuver), and (3) to construct a barrier of fascial tissue by suture of one of the aforementioned structures, notably the transversalis fascia, to Cooper's ligament.

The chief disadvantage of this inguinal approach is the possibility of a direct inguinal hernia occurring through the weakened wall of the

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10. Ochsner, A. J.: Ueber femorale Herniotomie, Deutsche klin.-therap. Wchnschr. **13**:1063-1071, 1906; J. A. M. A. **97**:751-754 (Sept. 8) 1906.

11. Waugh, R. L., and Hansfeld, K. F.: Femoral Hernia, Am. J. Surg. **58**: 73-77, 1942.

12. Bassini, E.: Nuovo metodo operativo per la cura radicale dell'ernia crurale, Padova, A. Draghi, 1893.

13. Lotheissen, G.: Zur Radikaloperation der Schenkelhernien, Centralbl. f. Chir. **25**:548-550, 1898.

inguinal canal. Another possible difficulty may be encountered, i. e., the proximity of the femoral vein; this can be avoided with care. McClure and Fallis [1] cannily indicate the wisdom and advantage of the inguinal approach when a wrong or doubtful diagnosis has been made. In 18 per cent of their cases of femoral hernia the preoperative diagnosis was inguinal hernia. Through the inguinal approach, either hernia can be successfully managed.

Moore [14] outlines a procedure for closing the femoral ring by suturing Poupart's ligament to Cooper's ligament either from below by femoral approach or through the inguinal approach. The fascia of the external oblique muscle is then sutured to the pectineal fascia from below. Fascial suture is an excellent method of assuring tissue union but is not routinely used. Gatch [15] has described a different way of handling complicated gangrenous intestine involved in femoral hernias by stages; the method was attended by a low mortality in his hands.

OPERATIVE FINDINGS

McNealy [8] and Jens [16] report on the contents of the femoral sacs in strangulated hernias in the femoral region, and a combination of their figures, which closely approximate each other, show that in 50 per cent of the cases small intestine alone is in the sac, in 25 per cent omentum and small intestine, in 20 per cent omentum alone and in the remaining cases nothing or else some other viscus, notably the appendix. Of the twelve strangulated hernias reported here, nine contained small intestine, two contained omentum and one contained small intestine and omentum. Fluid is frequently present, and in strangulation it is often bloody. The sac often presents a bluish, edematous appearance, and not infrequently the bluish color can be seen through the skin. The sac should be opened with care and the contents examined. When circulatory impairment of small intestine is present, judgment must be exercised as to the necessity for resection. Intestinal resection is accompanied with a high mortality, particularly when done through the femoral ring. The bowel may appear dusky and yet be viable. If there is serious doubt as to its viability and if the femoral ring does not allow delivery of a large loop, an abdominal incision should be made. Herrlin, Glaser and Lange [17] advocated injection of procaine hydrochloride about the vessels in the root of the mesentery in the loop affected. If

14. Moore, S. W.: Femoral Hernia, Surgery **13**:145-151, 1943.

15. Gatch, W. D., and Montgomery, W. F.: Treatment of External Hernias Containing Gangrenous Bowel: New Method Utilizing Miller-Abbott Tube, J. A. M. A. **129**:736-739 (Nov. 10) 1945.

16. Jens, J.: Strangulated Femoral Hernia, Lancet **1**:705-707, 1943.

17. Herrlin, J. O.; Glaser, S. T., and Lange, K.: New Methods of Determining Viability of Bowel, Arch. Surg. **45**:785-791 (Nov.) 1942.

the bowel is viable, the color will change rapidly from a dusky bluish red or black to red or pink. They regard this as a highly reliable guide for resection. The need for resection should be amply demonstrated, since reports in the literature show a mortality of 50 to 60 per cent.

End to end anastomosis is favored as the simplest method of resection. Two resections were necessary among the 19 cases reported (table 1), and end to end anastomosis in the ileum was employed. The intestinal anastomosis and the hernia repair were successful in both cases. A lengthy abdominal incision is neither necessary nor desirable, as the loop is easily found and can be delivered through a medium-sized wound without difficulty.

TABLE 1.—*Operations for Femoral Hernia in Nineteen Consecutive Cases from 1938 to 1946 Inclusive*

Patient	Age	Sex	Side	Operation	Follow-Up Period
			Strangulated Hernias		
O. N.	82	M	Left	Modified Bassini	Died after 6 years
J. G.	62	M	Right	Modified Bassini	8 years
R. A.	58	M	Right	Modified Bassini	Died after 7 years
J. E.	57	F	Right	Modified Bassini	7 years
C. N.	68	F	Left	Modified Bassini	Died after 5 years
A. S.	80	F	Right	Bassini, abdominal exploratory laparotomy	Died after 5 years
W. H.	70	M	Left	Bassini	5 years
C. P.	79	F	Right	Bassini, abdominal intestinal resection	Died after 4 years
O. A.	56	M	Right	Bassini, abdominal intestinal resection	3 years
C. L.	65	M	Left	McVay	2 years
L. W.	69	M	Right	McVay	3 months
P. N.	53	M	Right	McVay	4 months
			Nonstrangulated Hernias		
C. B.	43	F	Left	Bassini	8 years
J. T.	58	F	Left	Bassini	4 years
N. L.	57	F	Right	McVay	3 years
T. H.	30	F	Right	McVay	3 years
C. H.	73	F	Right	McVay	Died after 2 years
I. M.	55	F	Right	McVay	14 months
J. H.	68	M	Left	McVay	12 months

MORTALITY AND RECURRENCE

Death from femoral hernia is due to strangulation and occurs almost exclusively in instances in which the strangulated tissue was nonviable (table 2). It is readily apparent that to reduce the high mortality rate strangulated hernias must be operated on early and femoral hernia repaired when seen to prevent strangulation.

While there are hundreds of articles describing methods of operation and the odd viscera that have been found in the sacs, there are but a score that report the results of follow-up studies and the recurrence rates; some of these are not encouraging. It is particularly unfortunate that more specific data on recurrence rates are not available for the different types of operations. Ochsner,[10] Burrows [3] and Waugh [11] have given excellent accounts of the rates for simple ligation of the sac.

Apparently, when the Bassini operation is employed there is a recurrence rate of about 5 to 10 per cent. The latter figure is probably closer to the true one, since the cases must be followed at least two years. Fully one half of the recurrences after operation for femoral hernias take place after one year, in contrast to recurrences after operation for inguinal hernia, which usually take place within a year.

From a study of the statistics on recurrence in table 3 and of the methods used the conclusion might be made that Socin's original method of simple but adequate ligation of the sac, practiced so successfully by Ochsner and, more recently, by Burrows and Waugh, may give the best results in the treatment of small femoral hernia with a small unde-

TABLE 2.—*Operative Mortality*

Investigator	Total Cases	Nonstrangulated Hernias		Strangulated Hernias		Resections, per Cent	Mortality, per Cent
		Number	Per Cent	Number	Per Cent		
Shelley	222	184	0.5	38	17	..	3
Dean, G. D.: Arch. Surg. 44:933-942 (May) 1942	222	191	1	31	32	..	5
Douglas	103	...	...	51	17	30	9
Jens	100	...	...	100	14	66	..
Moore	121	...	...	10	0	..	..
McNealy	173	...	...	173	23	60	..
Dunphy	211	160	...	51	20	50	5
McClure	90	69	...	21	25	60	4.4

TABLE 3.—*Recurrence in Femoral Hernia*

Investigator	Cases in Which Operation Was Performed	Followed Cases	Recurrence, per Cent
Shelley	222	156	5
Dean	191 nonstrangulated,	...	12
	21 strangulated	...	23
Moore	121	...	4 (After reoperation; 1%)
McClure-Fallis	90	90	7.8
Waugh	12	10	0

formed femoral ring. The observation might be carried somewhat further and the Bassini operation likened to this method of simple ligation in conjunction with the suture of structures which are admittedly rather poor aids to firm healing and which at best close only the femoral canal. The recurrences following the Bassini operation are no doubt due to inadequate treatment of the sac or to the fact that it was carried out on patients having a large or deformed ring. These patients require a suture barrier across this opening, such as is accomplished by the inguinal approach.

There were no recurrences among the 19 cases reported here.

SUMMARY AND CONCLUSIONS

1. Femoral hernia occurs in patients in the later decades of life. Strangulation occurs frequently, with a relatively high mortality.

2. Renewed interest and anatomicosurgical studies of Cooper's ligament, or the superior pubic ligament, have led to improved methods of effectively closing the femoral ring as well as the weak area in direct inguinal hernia.

3. Low intestinal obstruction is frequently due to femoral hernia which of itself presents no symptoms or signs. The routine examination of the hernial rings is a necessity, and femoral hernia, if present, can usually be considered the cause.

4. Nineteen cases of femoral hernia are reported; twelve of the hernias were strangulated, two requiring abdominal intestinal resection. There were no deaths and no recurrences. Fifteen of the cases were followed for two years or longer and 17 for over one year.

NOTE.—Since the reading of this report 6 additional patients have been operated on for the repair of femoral hernias, bringing the total number to 25. In each of these 6 cases the Cooper ligament technic was used in the repair of the hernia, in keeping with the McVey procedure. The ages of these 6 patients varied from 25 to 80 years, and 4 of them were women and 2 men. In 2 patients the hernia was strangulated. One of these, a woman of 50, had been acutely ill for three days, and repeated reduction of the hernia had been attempted by her physician before she was brought to the hospital. At operation the ileum was gangrenous and had perforated. Through an abdominal approach a loop of gangrenous ileum was resected and an anastomosis performed. Seven days after operation the patient died of mesenteric thrombosis. This case serves to emphasize the dangers of strangulated femoral hernia and particularly the necessity for handling the tissues gently if reduction of the hernia is attempted. Of the 25 cases the hernias were strangulated in 14, were recurrent in 2 and uncomplicated in 9. One death occurred among 3 patients requiring intestinal anastomosis. No recurrences were found in the follow-up of the remaining 24 patients.

CONGENITAL DIAPHRAGMATIC HERNIA

Anatomic and Surgical Importance of the Left Triangular Ligament of the Liver

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CHICAGO

DIAPHRAGMATIC hernia is not an uncommon lesion in the newborn, and since it can be satisfactorily and permanently cured by proper surgical procedures, its early recognition bears reemphasis. Although an extensive literature is available on the general subject of diaphragmatic hernia, too little attention has been given to the condition during the first few days or weeks of life. Sudden deaths at birth or shortly after are usually attributed to a congenital cardiac lesion or to an enlarged thymus; it would be well if congenital diaphragmatic hernia would be considered and the diagnosis ruled out in such instances. Grulee [1] has stressed the fact that it should be suspected when a diagnosis of "supposed dextrocardia" is made.

Little or no mention has been made of the anatomic and surgical importance of the left triangular ligament of the liver as it pertains to exposure and surgical repair of congenital diaphragmatic hernia. A brief review of the structure of this ligament will aid in the clarification of its surgical importance. The falciform ligament consists of two layers of peritoneum (fig. 1). At the back part of the upper surface of the liver, the left layer of this ligament turns to the left and forms the upper layer of the left triangular ligament. This passes from the upper surface of the left lobe of the liver to the diaphragm. At the left end of the left triangular ligament that layer turns to the right as the lower layer of the left triangular ligament, and at the upper end of the fissure for the ligamentum venosum it becomes continuous with the lesser omentum. The left triangular ligament is a rather extensive peritoneal fold which can be clamped between two fingers. The right triangular ligament is not so well marked. Its two layers diverge at once as the upper and lower layers of the coronary ligament which limit the bare area on the back of the liver above and below. The upper layer of the coronary ligament is reflected from the right

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1. Grulee, C. G.: Hirschsprung's Disease, Internat. Clin. **2**:100, 1915.

lobe of the liver onto the diaphragm; the lower layer is reflected onto the right kidney as the hepatorenal ligament. When the bloodless left triangular ligament is cut the esophageal hiatus and the diaphragmatic hernia can be seen. This maneuver permits mobilization of the left lobe of the liver to the right. When one fails to take advantage of this, it becomes extremely difficult to visualize the hernial ring since the liver covers it. This makes reduction of contents and repair extremely difficult.

Morgagni in 1769 wrote the first monograph on the subject, although Ambroise Paré reported 2 traumatic cases in 1610. A marked variance of the published reports makes it difficult to estimate the incidence of the condition. In 1920 MacMillan [2] found only 3 cases in the course

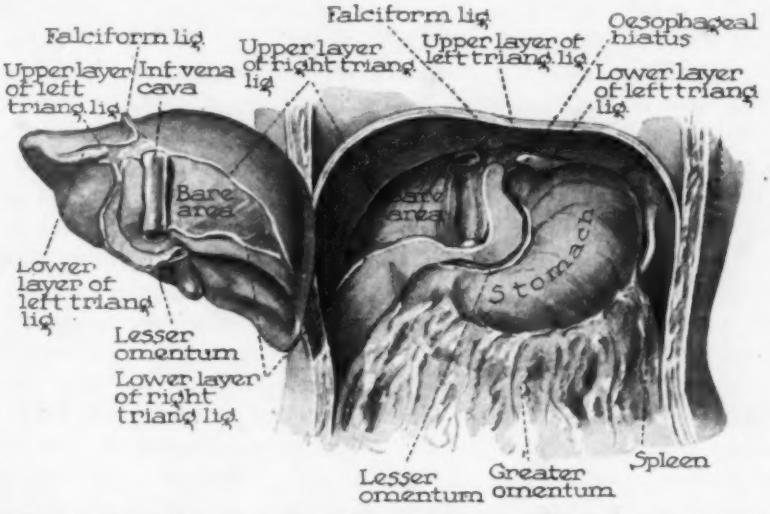


Fig. 1.—The ligaments of the liver. The liver has been displaced from the peritoneal cavity so that its ligamentous attachments may be seen. This illustration depicts a view of the posterior aspect of the liver and an anterior view of the posterior parietal wall to which the liver is attached. It should be noted that by cutting the left triangular ligament the left lobe of the liver can be displaced to the right, resulting in adequate exposure of the esophageal hiatus.

of fifteen thousand roentgenologic examinations, and in 1924 Pancoast and Boles [3] reported 16 cases discovered in nine thousand series of gastrointestinal roentgenograms. Bradley [4] in 1930 quoted figures from the Mayo Clinic which gave the number of patients with the condition

2. MacMillan, A. S.: Diaphragmatic Hernia, Am. J. Roentgenol. **7**:143, 1920.

3. Pancoast, H. K., and Boles, R. E.: Nontraumatic Diaphragmatic Hernia: Clinical and Roentgenologic Studies in Fifteen Cases, Arch. Int. Med. **38**:633 (Nov.) 1926.

4. Bradley, W. N.; Zulich, J. D., and McKee, E. E.: Congenital Diaphragmatic Hernia: Preliminary Report of a Case, Arch. Pediat. **47**:384, 1930.

as 1 in 18,000 admitted. After a thorough search of the literature, Truesdale [5] published data in May of 1936, stating that comparatively few cases have been reported. He attributed the apparent rarity to difficulty of recognition rather than to infrequency. He stated that only 21 patients under 10 years of age had been operated on before 1931 and that in this group the mortality was 41 per cent. In 1931 Hedblom [6] collected 1,435 cases from the literature. In 1933 Harrington [7] revealed that they had five times as many cases at the Mayo Clinic during the preceding eight years as they had had during the previous twenty-four years.

EMBRYOLOGIC DEVELOPMENT

The embryologic development of the diaphragm is complicated because the diaphragmatic elements are derived from several sources. The anterior, lateral and central parts which make up the greater portion are formed from the transverse septum and the fused ventral mesentery. The posterolateral part is formed by the fusion of the dorsal mesentery, and the mesoderm is derived from the receding wolffian body and the pleuroperitoneal membrane. Some embryologists divide diaphragmatic development into three stages: In the first stage, the celomic space is a continuous one until the growth of the organs divide it into pericardial, pleural and peritoneal cavities. The septum transversum forms the ventral part of the diaphragm; the pericardial cavity lies ventral to it. Since this septum is confined to the ventral half of the embryo, the pleural space remains continuous with the peritoneal cavity posteriorly. This resulting continuity is the pleuroperitoneal canal and the developing esophagus, stomach and its mesentery pass through it. Should the development proceed only to this point, the abdominal organs would have a direct route into the thorax. A hernia occurring during this stage would have no sac. In the second stage, the pleuroperitoneal canal becomes obliterated by the proliferation of the primitive dorsal mesentery of the stomach and esophagus. The pleural and peritoneal cavities are now fully formed, and if a herniation should occur during this phase, a sac would be present. During the third or final phase, the phrenic nerve with a muscle mass invades the diaphragm. This muscle replaces the membranous structures; the last area to be muscularized is the dorsal part. If the ingrowth of muscle is incomplete, the posterior part will be closed only by pleura and peritoneum, and in this instance the crura of

5. Truesdale, P. E.: Diaphragmatic Hernia: Varieties and Surgical Treatment of the Hiatus Type, Am. J. Surg. **32**:204, 1936.

6. Hedblom, C. A.: Diaphragmatic Hernia, Ann. Surg. **94**:776, 1931.

7. Harrington, S. W.: Diaphragmatic Hernia: Symptoms and Surgical Treatment in Sixty Cases, J. A. M. A. **101**:987 (Sept. 23) 1933.

the diaphragm which form the back of the muscle will be absent in whole or in part.

CLASSIFICATION

Although many classifications have been utilized, it seems wise to use one which is of practical value. According to most authors, congenital diaphragmatic hernias occur at the following sites:

1. In the posterolateral portions along the embryonic pleuroperitoneal canal (foramen of Bochdalek). This is the most common site, and hernias appearing here are about four times more common on the left than on the right.

2. Through the esophageal hiatus. This is the type which is usually due to a lack of development of one or both of the diaphragmatic crura. The case which I am reporting is of this type.

3. In the retrosternal area (foramen of Morgagni). These are herniations through the retrosternal attachments of the diaphragm and are the least common.

Herniations through the aortic or vena caval openings in the diaphragm have not been reported.

When a diaphragmatic hernia has a sac, its superior border is limited. When no sac is present, the involved pleural cavity is filled with collapsed lung, stomach, small and large bowel and either the spleen or the liver, depending on which side the hernia occurs on.

SYMPTOMATOLOGY AND DIAGNOSIS

Congenital diaphragmatic hernia may give symptoms referable to the three systems usually involved, namely, respiratory, circulatory and digestive. These result from mechanical interference with the functions of the viscera, and the severity of the symptoms depends on the number of organs displaced. Any newborn infant who continually displays cyanosis, dyspnea or vomiting or any combination of these should be suspected of having a diaphragmatic hernia. The symptoms vary to a considerable degree, and many patients may even be symptom free. At times the hernia is only discovered during the course of a routine roentgenologic examination. Greenwald and Steiner [8] stated that cyanosis is the most constant sign. This may be present immediately after delivery, or it may be transient and appear during crying or nursing. Occasionally it can be relieved by turning the infant so that the side of the hernia faces downward. Respiratory difficulties vary from a spontaneous failure to breathe, with subsequent death at birth, to the insignificant yet suggestive observations of a peculiar weak cry. Many have mentioned the diagnostic value of the latter. Regarding

8. Greenwald, H. M., and Steiner, M.: Diaphragmatic Hernia in Infancy and in Childhood, Am. J. Dis. Child. **38**:361 (Aug.) 1929.

the gastrointestinal symptoms, Harrington [7] stated that the vague dyspeptic symptoms are not in proportion to the severity of the lesion. Difficulties begin when, owing to an overloaded bowel or stomach, a state of increasing distention of the intestine occurs. As a result of this the increased thoracic pressure impedes the action of the heart and lungs. Once the intestines have passed into the chest, the negative thoracic pressure prevents and hinders spontaneous reduction. Vomiting may follow most of the feedings, or it may be an occasional and insignificant complaint. In patients surviving for one or two months some loss of weight is usually noted.

Physical examination usually reveals increased pulse and respiratory rates. On inspection, the affected side of the chest is less mobile and the abdomen somewhat scaphoid. Percussion of the chest on the involved side may reveal dulness or tympany, depending on which viscera are present. Dulness is usually present on the uninvolved side. Auscultation may transmit gurgling sounds instead of breath sounds. When the latter are heard, however, they are extremely distant. The heart sounds are heard on the unaffected side.

Diagnosis is always confirmed by roentgenologic examination. This procedure affords a fairly accurate method of estimating what abdominal viscera are present in the thoracic cavity, and even fluoroscopic examination without the use of contrast mediums supplies the necessary information. Although the use of barium is supposed to increase the incidence of vomiting and of aspiration pneumonia, it may be employed if the mixture is kept thin.

REPORT OF A CASE

On Nov. 30, 1946, a 7 week old white female infant (J. P.) was admitted to the American Hospital, with a history of cyanosis and respiratory difficulty after feeding. The child was the first born, and there were no obstetric difficulties or complications. The mother stated that each time the baby would feed she would draw her legs up and cry, being apparently in pain. She further stated that after a feeding of 2 or 3 ounces (56 to 85 Gm.) the child would become cyanotic and have a convulsive seizure, which would be followed by vomiting. Although these symptoms were apparent shortly after birth, they became more severe three weeks prior to admission to the hospital. The patient had occasional hard stools. Physical examination revealed an alert and active infant weighing 8 pounds and 2 ounces (3.6 Kg.). The skin showed a moderate cyanosis, which deepened when the baby cried; hydration was good. The scalp was clear, and the anterior fontanel measured 1 by 1 cm. The eyes, nose and throat were normal, and there was no torsion of the neck. The chest was apparently symmetric and full; expansion was greater on the right side. On percussion of the lungs, the right was found to be hyperresonant throughout and the left was resonant in the apex; tympany was present on the left side from the second to the fifth intercostal space, with dulness below. The left border of the heart was at the right midclavicular line. Breath sounds were high pitched on the right side and absent on the left. Heart tones were normal. The abdomen was scaphoid and firm;

the liver and spleen were not felt. Only a few short peristaltic sounds were heard. The extremities were normal, as also were the reflexes. Laboratory examination revealed normal urine, 4,550,000 erythrocytes and 9,750 leukocytes; the differential leukocyte count was normal. The hemoglobin content was 14 Gm. per hundred cubic centimeters, or 90 per cent. Roentgenologic examination revealed a congenital diaphragmatic hernia on the left side. The left thoracic cavity contained stomach and small and large bowel. The spleen, which was also present in the thorax, was not visualized in the roentgenogram (fig. 2). The heart and mediastinum were pushed to the right. Only a little pulmonary shadow was seen on the right and none on the left.

TREATMENT

The treatment is surgical. Expectant or medical therapy is futile and wastes much valuable time. Waiting until the child is older or stronger may cause loss of life, and hence surgical intervention should

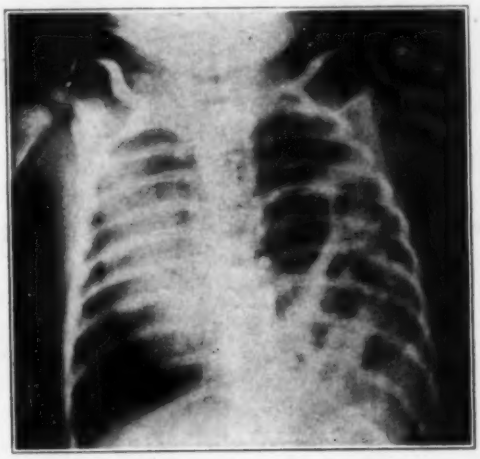


Fig. 2.—Roentgenogram revealing congenital diaphragmatic hernia on the left side. Gas-filled stomach and large and small bowel can be seen in the left thoracic cavity; the heart and mediastinal structures have been pushed to the right.

be contemplated as soon as the diagnosis is made. Ladd and Gross [9] stated that an operation performed early is advantageous since the surgeon has the advantage of dealing with an intestinal tract which is not distended or enlarged and can be replaced within the abdominal cavity. A disadvantage was present in the case reported here in that the patient was 7 weeks old.

Preoperative measures are aimed at deflation of the alimentary tract, maintenance of proper hydration and prophylactic chemotherapy.

Anesthesia plays a vital part in the successful outcome of such conditions. Regardless of the anesthetic agent employed, positive

9. Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941.

pressure anesthesia must be available at all times. Ochsner, DeBakey and Murray [10] reported a case of congenital diaphragmatic hernia in which there was complete absence of the anterior mediastinum. This resulted in a single pleural cavity. Others have reported such cases and stress the fact that the opening into one pleural cavity would permit atmospheric pressure to collapse both lungs. Positive pressure anesthesia becomes imperative. Cyclopropane and ether are the agents most commonly used.

Some authorities advocate preliminary crushing of the phrenic nerve, but in the case herein reported the movements of the diaphragm did not hinder the placing of the sutures.

The technic of operative repair and exposure in diaphragmatic hernias is somewhat different in infants than in adults. Although a transthoracic approach is probably best in adults, the abdominal route is far superior in infants. This is true because adhesions between intestines and thoracic structures are rare in infancy, and it is much easier to reduce the abdominal contents from below than to push them down from above. Furthermore, it is usually impossible to reduce such a hernia from the thoracic side because the abdominal cavity is too small to accommodate the viscera. The abdominal incisions preferred are either vertical or subcostal. In the case under discussion a long muscle-splitting incision over the rectus was used, which extended from the left of the xiphoid to well below the umbilicus (fig. 3 *B*). When the peritoneal cavity was entered the paucity of viscera was immediately noted; these consisted of liver, a few coils of ileum, sigmoid and rectum. The pylorus entered the hernial hiatus and the descending colon filled the rest of the hernial orifice. As has been stated, the practical importance of the left triangular ligament of the liver has not been stressed. The nature of this bloodless peritoneal fold must be understood in order to acquire proper exposure of the hernial orifice. When this structure is cut the left lobe of the liver can be retracted upward and to the right; this maneuver immediately reveals the hernial hiatus and exposes it completely (fig. 3 *C*). Should this step be overlooked, much unnecessary trauma may result, making repair difficult and at times inadequate. It is unnecessary to resuture this ligament, since merely replacing the left lobe of the liver results in its immediate sealing of itself into place. This has been personally observed at autopsy. A rubber catheter is introduced through the hernial ring to permit air to enter the thorax (fig. 3 *D*). If one fails to do this, the negative pressure within the thoracic cavity will suck back the abdominal viscera as fast as the surgeon attempts to reduce them. Some authors advocate withdrawal

10. Ochsner, A.; DeBakey, M., and Murray, S.: Absence of the Anterior Mediastinum with Report of a Case Associated with Congenital Diaphragmatic Hernia, Surgery 6:915, 1939.

of the abdominal viscera in a definite order. However, during the course of reduction, the viscera seem to reduce themselves by a continuity all their own.

After their reduction the viscera should be placed outside of the abdominal cavity, opposite the side of the hernial orifice, and wrapped in warm moist gauze packs. In the case herein reported there was no

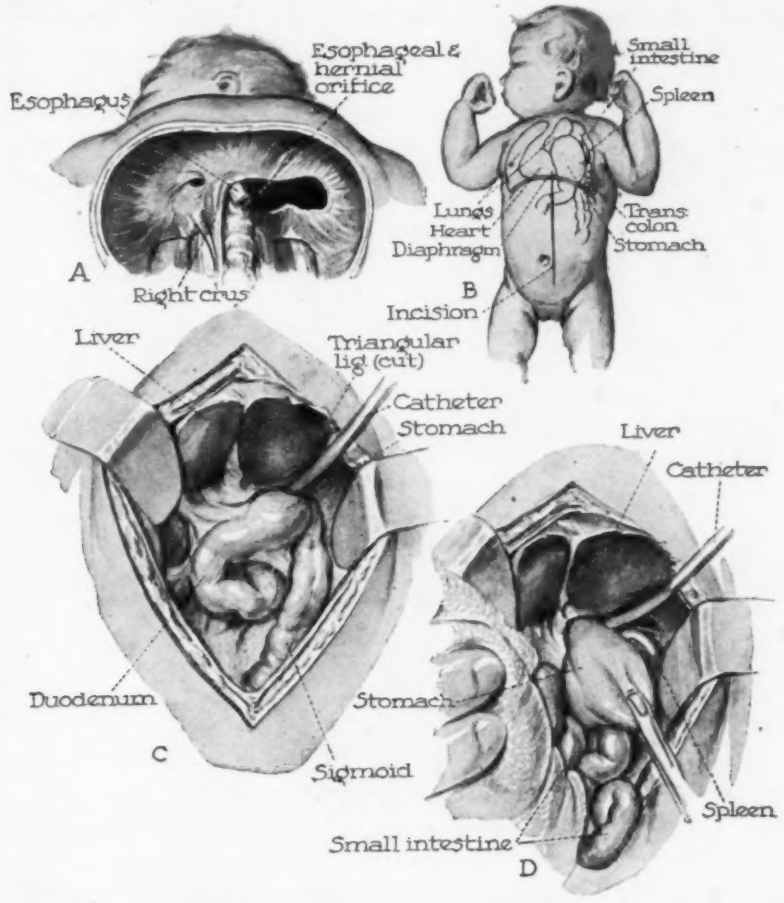
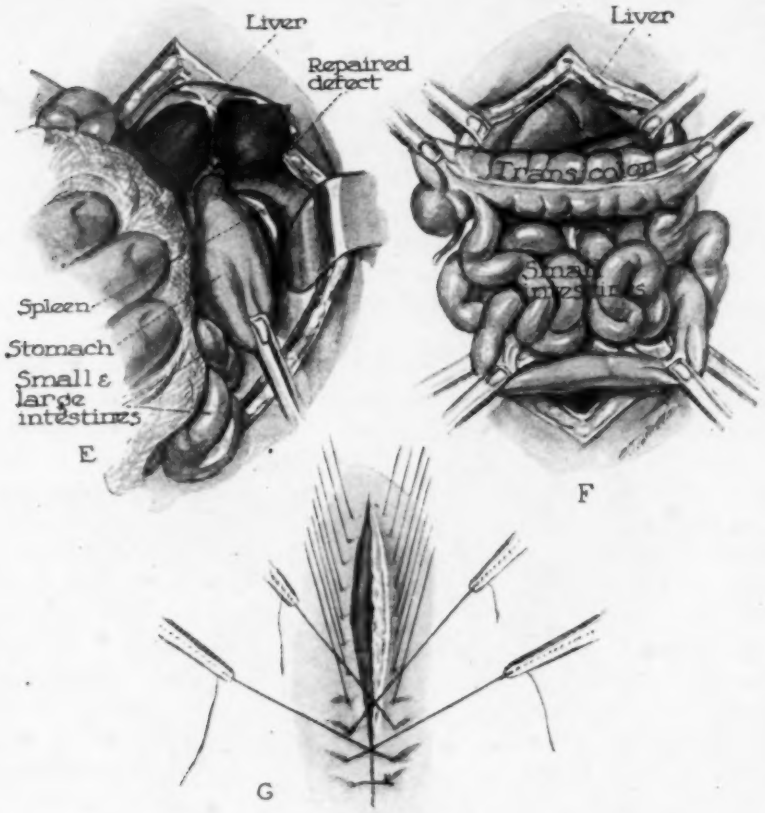


Fig. 3.—Surgical repair of congenital diaphragmatic hernia. *A*, the hernial hiatus in this case included the esophageal orifice and most of the left leaf of the diaphragm. Such a defect is brought about by the nondevelopment of the left diaphragmatic crus. *B*, the herniated abdominal viscera are shown. The incision extends from the left side of the xiphoid to well below the umbilicus. *C*, the left triangular ligament has been cut, the liver retracted and the hernial hiatus exposed. *D*, a rubber catheter is introduced through the hernial ring to permit air to enter the thorax. This equalizes the negative pressure within the thoracic cavity,

hernial sac; however, should such a sac be present, it can be incised and air permitted to enter the pleural cavity. The sac is then delivered

into the abdomen, excised and sutured as part of the closure of the hernial ring. At this stage in the operation the type of diaphragmatic defect is determined. As has been stated, the most common is the so-called foramen of Bochdalek, but in this case the esophageal hiatus was found to be included. This was due to a lack of development of the left diaphragmatic crus (fig. 3 *A*). Many methods of repairing the hernial opening have been described. Denudation of the edge of



aiding in the reduction of the herniated viscera. *E*, simple closure with interrupted nonabsorbable sutures. *F*, after reduction and repair, the viscera are replaced into the abdominal cavity. *G*, closure is accomplished by using heavy interrupted cotton sutures which include all layers of the anterior abdominal wall and are placed one-quarter of an inch (0.64 cm.) apart. These are elevated, and the lowermost suture is tied while the one next to it is crossed. By this method tension is relieved and healing takes place without the development of an incisional hernia.

the hernial ring followed by approximation with two rows of interrupted sutures is the one most commonly used. I have found that a

simple closure with interrupted cotton sutures suffices. Care was taken not to constrict the newly formed esophageal hiatus (fig. 3 *E*).

The viscera which have been removed during the herniorrhaphy are replaced into the abdominal cavity and an abdominal closure planned (fig. 3 *F*). Since the abdomen is underdeveloped, the surgeon usually finds it difficult to accomplish a layer by layer closure. If the closure is forced, it results in great tension on the edges of the wound, increased intra-abdominal pressure and respiratory embarrassment. Some advocate undercutting the skin to allow it to slide and then closing the subcutaneous fascia and skin only. This results in an incisional hernia, which requires later repair. I have utilized the same type of closure that is used in postoperative dehiscence of a wound. This is accomplished in the following way: Heavy interrupted cotton sutures which include all layers of the anterior abdominal wall are placed about one quarter of an inch (0.64 cm.) apart. After their placement, they are elevated and the lowermost suture is tied while the one next to it is crossed (fig. 3 *G*). By this method, tension is taken off the suture being tied. It is easy to accomplish and avoids an incisional hernia.

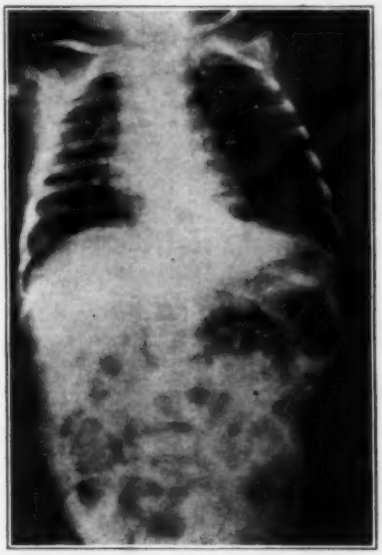


Fig. 4.—Roentgenogram taken twenty-four hours after operation. The abdominal viscera have been returned to the abdominal cavity; the left lung is beginning to reexpand, and the heart has assumed its normal position.

The postoperative care can cause success or failure. Blood transfusions should be given after the operation, but since the right side of the heart may be embarrassed in infants with this condition the transfusion must be adequate yet small. Usually 125 cc. will suffice. Oxygen is given postoperatively in 90 to 95 per cent concentration by means

of a tent; its use should be continued for three to five days. This not only makes breathing easier but helps to keep the intestines deflated. Feeding by mouth is started early, but if this is impossible and if caloric requirements cannot be fulfilled then parenteral routes are used. Chemotherapy is utilized and vitamin balance maintained. Postoperatively roentgenograms are taken frequently to determine the status of the reexpanding lung and the sutured diaphragm and to detect the presence or absence of pleural fluid. It was interesting to note that in the case discussed no fluid could be demonstrated at any time in the pleural cavity and that the reexpansion of the lung progressed favorably (fig. 4). The child left the hospital two weeks after operation and made an uneventful recovery.

SUMMARY

A case of congenital diaphragmatic hernia due to nondevelopment of the left crus of the diaphragm in a 7 week old infant, with surgical repair and recovery, is reported.

The importance of the left triangular ligament of the liver as pertaining to exposure is stressed.

A simple closure of the hernial defect with interrupted cotton sutures is described.

Utilization of through and through interrupted sutures which include all layers of the anterior abdominal wall may prevent the necessity of a second surgical procedure for the repair of an incisional hernia.

The important roles played by the anesthetist and the pediatrician are emphasized.

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ABSORPTION OF BLOOD FROM THE PERITONEAL CAVITY

CLAUDE R. SNEAD, M.D.
CHICAGO

THIS investigation was stimulated by the observation that surgeons differ concerning the advisability of removal of fluid blood and clots from the peritoneal cavity after intraperitoneal hemorrhage or operative procedures. The purpose of the experiments was to determine the fate of blood introduced into the peritoneal cavity, its possible usefulness as a substitute for transfusion on occasions when the usual methods are inapplicable or too difficult and its effect on the blood volume, hematocrit reading and hemoglobin and total protein levels. Additional observations were made to determine if the clinical leukocytosis so frequently observed after intraperitoneal hemorrhage is due to the hemorrhage alone or to the mere presence of blood within the peritoneal cavity. The response of the peritoneum itself to repeated intraperitoneal injections of autogenous blood was also determined.

A review of the literature concerning peritoneal absorption prior to 1923 was written by Cunningham.[1] The method of administering blood by the intraperitoneal route as a means of transfusion or as a preventive measure against peritonitis or adhesions has been studied by many investigators.[2] For example, Sparks and David [3] concluded that the presence of blood in the peritoneal cavity had no effect on the development of peritonitis, while Joseph [4] reported that blood in the peritoneal cavity after abdominal operations offered protection

From the Department of Surgery, University of Illinois College of Medicine.

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Presented before the meeting of the American College of Surgeons, Forum of Fundamental Surgical Problems, New York, Sept. 11, 1947.

1. Cunningham, R. S.: Physiology of the Serous Membranes, Physiol. Rev. **6**:242, 1926.

2. Siperstein, D. A., and Sansby, J. M.: Intraperitoneal Transfusion with Citrated Blood, Am. J. Dis. Child. **25**:107 (Feb.) 1923. Florey, H., and Witte, L. J.: Absorption of Blood from the Peritoneal Cavity, Lancet **1**:1323, 1928.

3. Sparks, J. P., and David, V. C.: Effect of Blood in the Peritoneal Cavity upon the Production of Peritonitis in Animals, Surg., Gynec. & Obst. **48**:780, 1929.

4. Joseph, E. G.: Use of Whole Blood as Means of Preventing Peritonitis and Adhesions, Ann. Surg. **111**:618, 1940.

against peritonitis and adhesions. Although experimental studies [5] have shown that one or two intraperitoneal injections of blood produced no significant peritoneal change, the response of the peritoneum to repeated injections of blood over a prolonged period needs further study.

An increase in hemoglobin and in the erythrocyte count has been reported previously after the injection of blood into the peritoneal cavity, whereas the response of the blood volume to hemoperitoneum has not been investigated.

EXPERIMENT I: EFFECT OF ANTICOAGULANT ON PERITONEAL ABSORPTION OF BLOOD

Procedure.—In 10 adult dogs amounts of blood varying from 60 to 280 cc. were removed by arterial puncture. In 5 dogs the blood was

TABLE 1.—*Intraperitoneal Injection of Autogenous Blood Without Anticoagulant*

Dog	Body Weight, Kg.	Blood Injected, Cc.	Time of Laparotomy After Injection of Blood	Findings
1	10.4	140	24 hr.	Injected peritoneum, mesentery and omentum; clot 2.5 by 8 cm. within omentum
		Reoperated on	15 days	Clots absorbed; peritoneum normal; small bowel adherent to scar; no other adhesions
2	14.5	170	4 days	Six clots 0.5 to 2 cm. in size in omentum
		Reoperated on	18 days	Clots absorbed; peritoneum normal; three adhesions between omentum and mesentery
3	3.1	110	9 days	Peritoneum normal; no clots or adhesions
4	5	100	10 days	Peritoneum normal; no clots or adhesions
5	6	130	15 days	Peritoneum normal; no clots or adhesions

immediately introduced into the peritoneal cavity. For the remaining dogs the blood was citrated before peritoneal injection, 10 cc. of a 5 per cent solution of sodium citrate being used per hundred cubic centimeters of blood. At intervals thereafter, laparotomy was performed on all dogs to determine the fate of the injected blood.

Results.—Examination of table 1 indicates that in 2 dogs receiving injections of uncitrated blood and operated on within four days clots were still present within the peritoneal cavity, while in dogs examined after nine days no residual clots could be found. In no instance was liquid blood observed in the peritoneal cavity at the time of operation.

5. Darigo, A.: Experimental Study of Hemoperitoneum and Its Influence on Erythrocyte Count and Rate of Production of Adhesions, Policlinico (sez. chir.) **47**:15, 1940.

Table 2 demonstrates that only minute clots and fibrinous strands appeared when citrated blood was injected intraperitoneally. The citrated blood was completely absorbed within twenty-four hours.

EXPERIMENT II: EFFECT OF HEMOPERITONEUM ON THE VOLUME AND ON SOME CONSTITUENTS OF CIRCULATING BLOOD

Procedure.—To determine the effect of loss of blood and replacement of the lost blood intraperitoneally, 17 normal dogs weighing from 10 to 18 Kg. were divided into three groups. The first group of 5 animals served as controls. In the second group of 5 dogs 36 cc. of blood per kilogram of body weight was removed by femoral puncture,

TABLE 2.—*Intraperitoneal Injection of Citrated Autogenous Blood*

Dog	Body Weight, Kg.	Blood Injected, Cc.	Time of Laparotomy After Injection of Blood	Findings
6	5.4	108	3½ hr.	Peritoneum injected- approximately 50 cc. of blood; no clots
7	3.1	62	22 hr.	Peritoneum and omentum injected; omentum contained many clots 5 to 10 mm. in size; one fibrinous strand 3 cm. in length between omentum and mesentery; blood absorbed
8	8.1	162	28 hr.	Omental fibrinous strand 4 by 2 mm.; omentum injected; blood absorbed
9	10	200	45 hr.	Omentum injeected and contained three clots 5 to 10 mm. in size; blood absorbed
10	14	280	48 hr.	Approximately 20 cc. of blood-tinged fluid; two areas of fibrin deposit 5 to 10 mm. on serosa of small bowel; omentum injected and held two clots 1 to 2 cm. in size

the period of bleeding lasting three to thirty minutes. The third group of 7 dogs was bled similarly, and the blood withdrawn was citrated and preserved in the refrigerator. Twenty-four hours after withdrawal the blood was returned to the dogs by peritoneal injection. Sterile technic was used throughout the experiment.

For all animals, determinations of the circulating plasma volume, hematocrit reading, hemoglobin content, total protein level and white blood cell count were made before the withdrawal of blood, twenty-four hours after and at six, twelve, eighteen, twenty-four and forty-eight hours after peritoneal injection.

Plasma volumes were determined by the dye method,[6] with the use of Evans blue (T-1824). After an initial sample of arterial blood was

6. Gregerson, M. I.: A Practical Method for the Determination of Blood Volume with the Dye T 1824, J. Lab. & Clin. Med. **29**:1266, 1944.

withdrawn, 10 cc. of a 0.2 per cent solution of the dye was injected intravenously with a calibrated syringe. Ten minutes later another sample of blood was withdrawn and the amount of dye present estimated colorimetrically in a chamber 10 mm. in depth, a Cenco-Sheard Sanford "photelometer" with a red filter being used. Samples showing hemolysis were discarded. Any residual dye from a previous determination was taken into consideration by using the sample of blood in the colorimetric standard withdrawn before injection. From the figure obtained for plasma volume the total blood volume was calculated by use of the following formula:

$$\text{Circulating volume} = \frac{\text{plasma volume} \times 100}{100 - (0.96 \times \text{hematocrit value})}$$

The value 0.96 represents the correction factor for plasma trapped between the cells.

The hematocrit reading was determined with a Wintrobe hematocrit tube by centrifuging for fifteen minutes at 3,000 revolutions per minute. The hemoglobin content was estimated with the "photelometer," and the leukocyte count was done by the usual clinical laboratory methods. The total serum protein was determined by the Hoppe-Goetz [7] modification of the Looney-Walsh [8] turbidometric method.

Results.—Since all these determinations were made seven times during the course of the experiment, with withdrawal of 30 cc. of blood each time, a total of 210 cc. of blood was withdrawn from each dog for laboratory determinations.

The average normal blood volume (fig. 1) per kilogram of body weight for the three groups of dogs was found to be between 98 and 104 cc. Twenty-four hours after hemorrhage of 36 cc. of blood per kilogram an abrupt fall in total blood volume was apparent (curves *B* and *C*). A rise in blood volume (curve *B*) began six hours after intraperitoneal injection of blood and continued until the observation at the end of forty-eight hours, with the maximum rise occurring at twenty-four hours. In those animals in which the blood was not returned (curve *C*) a progressive fall in circulating blood volume occurred throughout the experiment.

Twenty-four hours after hemorrhage of 36 cc. of blood per kilogram of body weight the average hematocrit value (fig. 2, curves *B* and *C*) was reduced from 45 to 27.5 per cent. After peritoneal injection of blood (curve *B*) the hematocrit value increased from 28 to 31 per cent at the end of forty-eight hours, while at the same time the value in

7. Hoppe, E., and Goetz, R.: Unpublished data.

8. Looney, J. M., and Walsh, A. J.: Determination of Globulin and Albumin in Blood Serum by Photoelectric Colorimeter, J. Biol. Chem. **130**:635, 1939.

dogs not receiving peritoneal injection and in the normal controls (curves *A* and *C*) decreased a total of 8 per cent.

Figure 3 (curves *B* and *C*) demonstrates an average loss of 5.5 Gm. of hemoglobin twenty-four hours after hemorrhage of 36 cc. per kilogram of body weight. The hemoglobin level rises (curve *B*) 1 Gm. forty-

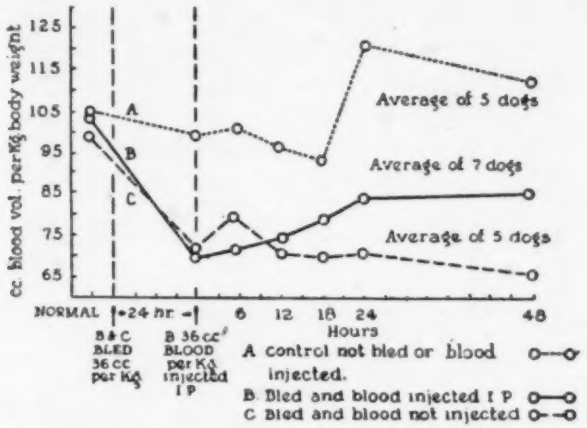

Fig. 1.—Fall in blood volume following hemorrhage in 12 dogs (curve *B* and *C*); there is a gradual rise in volume when blood is returned intraperitoneally (curve *B*), whereas the group in which blood was not returned intraperitoneally (curve *C*) showed a fall in blood volume consistent with the frequent withdrawal of 30 cc. of blood. The control group, having had blood withdrawn for chemical determinations only, is represented in curve *A*.

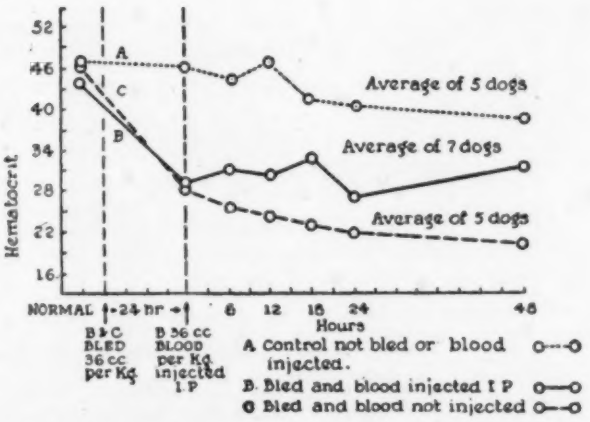

Fig. 2.—Fall in hematocrit reading following hemorrhage (curves *B* and *C*); there is a gradual rise when blood is returned intraperitoneally (curve *B*). The group in which blood was not returned intraperitoneally (curve *C*) showed a fall in hematocrit value consistent with the amount of blood withdrawn for determinations. Curve *A* shows the hematocrit readings in the control group, which was not subjected to hemorrhage or intraperitoneal injection.

eight hours after peritoneal injection, while the animals which had not received blood intraperitoneally (curve *C*) and the controls (curve *A*) lost 2 Gm. of hemoglobin during the same period.

Total serum protein levels twenty-four hours after hemorrhage (fig. 4, curves *B* and *C*) decreased about 1 Gm. per hundred cubic

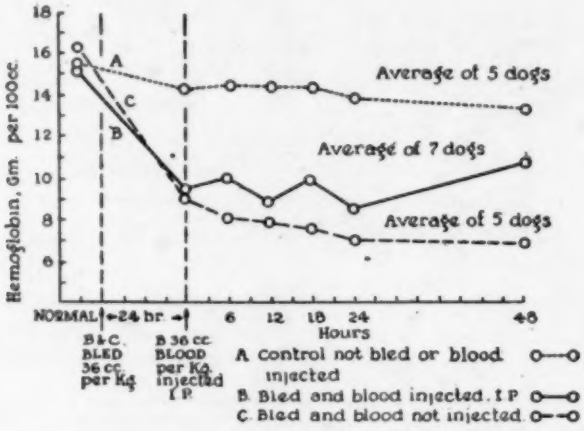

Fig. 3.—The hemoglobin value falls 6 Gm. per hundred cubic centimeters (curves *B* and *C*) after hemorrhage and rises 1 Gm. after intraperitoneal injection of blood (curve *B*). However, the animals in which there was no return of blood intraperitoneally (curve *C*) show an additional fall of 2 Gm. because of withdrawal of blood for laboratory determinations. Curve *A* represents determinations of hemoglobin in the control group.

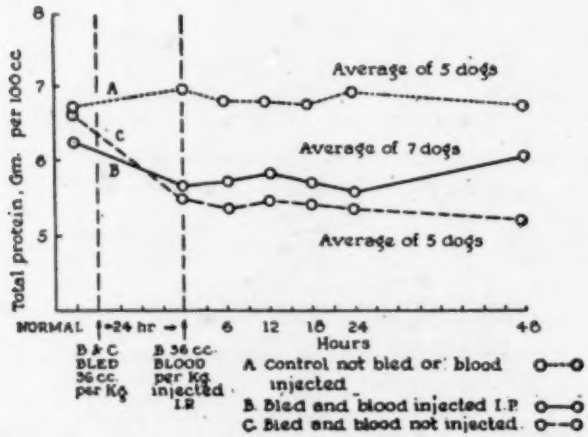

Fig. 4.—Total protein levels rose to near normal (curve *B*) after intraperitoneal injection of blood, while they remained stationary in the controls (curve *A*) and showed a slight but progressive fall in animals in which return of blood was not made.

centimeters. The group receiving intraperitoneal injection of blood (curve *B*) showed an increase of 0.5 Gm. from the time of injection until the observation at forty-eight hours, while at the same time the level in the two control groups (curves *A* and *C*) decreased 0.2 Gm. per hundred cubic centimeters of serum.

The average white blood cell count (fig. 5) before hemorrhage was 15,200. The effect of hemorrhage is shown by an increase of the leukocyte count to 20,600, and this elevation (curve *C*) was sustained throughout the experiment. In dogs in which the blood was returned by peritoneal injection (curve *B*) a sharp additional rise occurred within six hours, the level reaching a maximum of 36,000 at eighteen hours and falling forty-eight hours after intraperitoneal injection to 20,000.

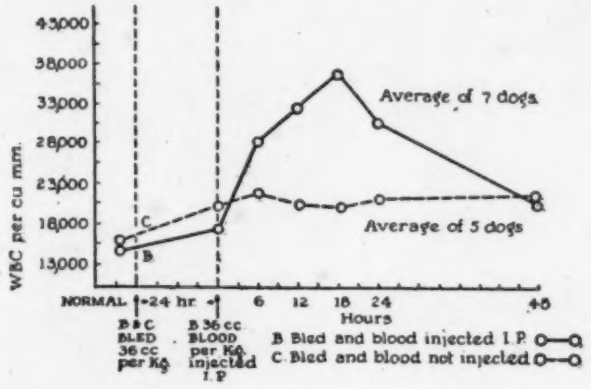

Fig. 5.—Slight rise of leukocyte count following hemorrhage in two groups of dogs and subsequent marked elevation (curve *B*) after return of blood intraperitoneally.

EXPERIMENT III: EFFECT OF REPEATED PERITONEAL INJECTIONS OF CITRATED AUTOGENOUS BLOOD ON MICROSCOPIC APPEARANCE OF THE PERITONEUM

Procedure.—To observe the effect of repeated intraperitoneal injection of blood, 15 cc. of blood was removed from 3 mature rabbits by cardiac puncture every third day. The blood was withdrawn by a syringe containing citrate and was immediately returned to the animal by intraperitoneal injection. After nine consecutive injections of 15 cc. of citrated blood and twenty-four hours after final injection, the rabbits were killed and microscopic sections of the peritoneum were made.

Results.—Postmortem examination of the rabbits disclosed thickening of the omentum and mesentery plus fibrinous clots and moderate injection of the omentum, with less injection of the remaining peritoneal structures. In no animals were adhesions encountered.

Microscopic studies of tissue from the omentum, mesentery, diaphragm, small bowel, liver and parietal peritoneum revealed some serosal fibrosis plus increased vascularity and mesothelial proliferation of the omentum. Nodular and filiform polypoid outgrowths of the serosa were found in the mesentery.

COMMENT

From the first experiment it becomes apparent that the rapidity of absorption of blood from the peritoneal cavity is influenced greatly by the presence of an anticoagulant. When blood with an anticoagulant (e. g., sodium citrate) was injected, absorption was rapid enough to

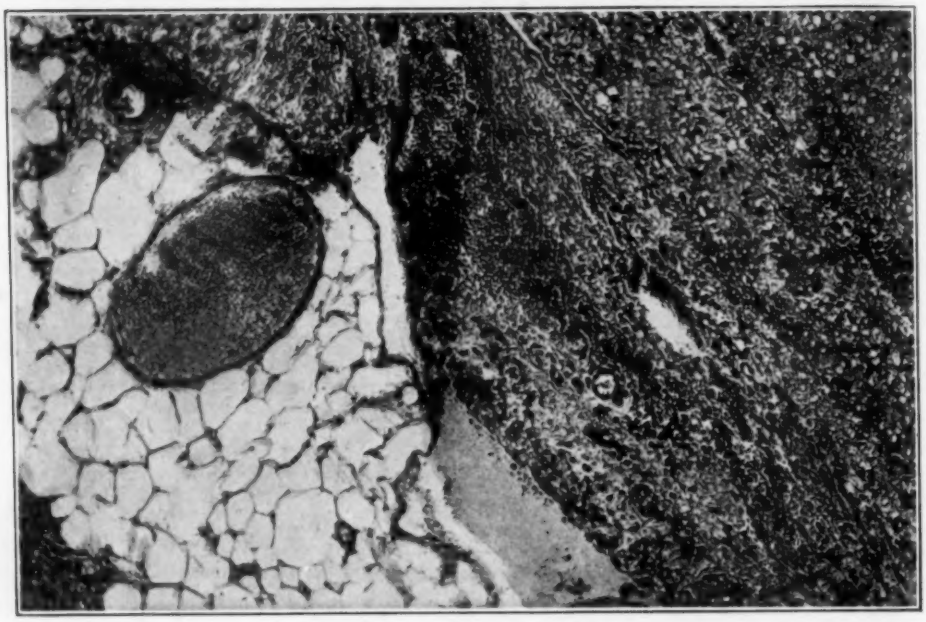

Fig. 6.—Microscopic section of omentum of rabbit showing moderate fibrosis and increased vascularity following nine intraperitoneal injections of 15 cc. of blood at three day intervals; × 110

indicate that intraperitoneal transfusion might be of some value as a method of replacement therapy. It is for that reason that a subsequent experiment was performed in which anticoagulant only was used.

The study of blood volume, plasma protein, hematocrit reading and hemoglobin demonstrates that blood in amounts between 250 and 600 cc. introduced into the peritoneal cavity finds its way into the circulating blood, as might be expected, but that the maximum increase in blood volume does not occur until twenty-four hours after injection. Though replacement of blood by way of the peritoneal cavity would be extremely

easy at the time of surgical operation, absorption is too slow to be of value in such conditions as postoperative shock. However, when rapid increase in blood volume is not mandatory intraperitoneal administration appears to be safe and satisfactory; it produces no serious pathologic changes within the peritoneal cavity.

The withdrawal of 30 cc. of blood for each series of laboratory determinations and the possibility[9] that anemic and hypoproteinemic dogs destroy red blood cells in the peritoneal cavity to replace plasma protein may explain the failure of the blood volume, hematocrit reading and hemoglobin levels to return to normal forty-eight hours after injection of blood intraperitoneally. The dogs in the experiment under discussion were moderately anemic and hypoproteinemic after hemorrhage of 36 cc. of blood per kilogram of body weight, and the rise of the total protein levels to normal after intraperitoneal injection of blood may have been due to destruction of red blood cells in the peritoneal cavity.

In the 17 animals receiving intraperitoneal injection of blood (experiments I and II) no immediate reaction or evidence of hemoglobinuria was observed. Study of the response of the leukocyte count definitely indicates that leukocytosis in clinical intraperitoneal hemorrhage is in small part due to hemorrhage. The introduction of blood into the peritoneal cavity[10] results in striking elevation of the white blood cell count, with almost equally rapid decline following absorption. The reason hemoperitoneum produces a high leukocyte count is at present obscure, but it is at least evident that hemorrhage itself is not a sufficient answer to the problem. In consideration of the postoperative peritoneal administration of blood, one must be cognizant of possible masking of the signs of postoperative complications by the leukocytosis which is secondary to peritoneal injection of blood.

From a study of experiment III it becomes apparent that repeated injections of citrated autogenous blood into the peritoneal cavity will provoke a mild fibrosis and thickening of the peritoneum. It also was observed that of the structures within the peritoneal cavity the mesentery, omentum and diaphragm were effected pathologically to a greater degree than were the other organs. In spite of mesothelial proliferation, peritoneal fibrosis and thickening, no adhesions were produced, which in my opinion indicates that the microscopic changes observed were not of great pathologic significance.

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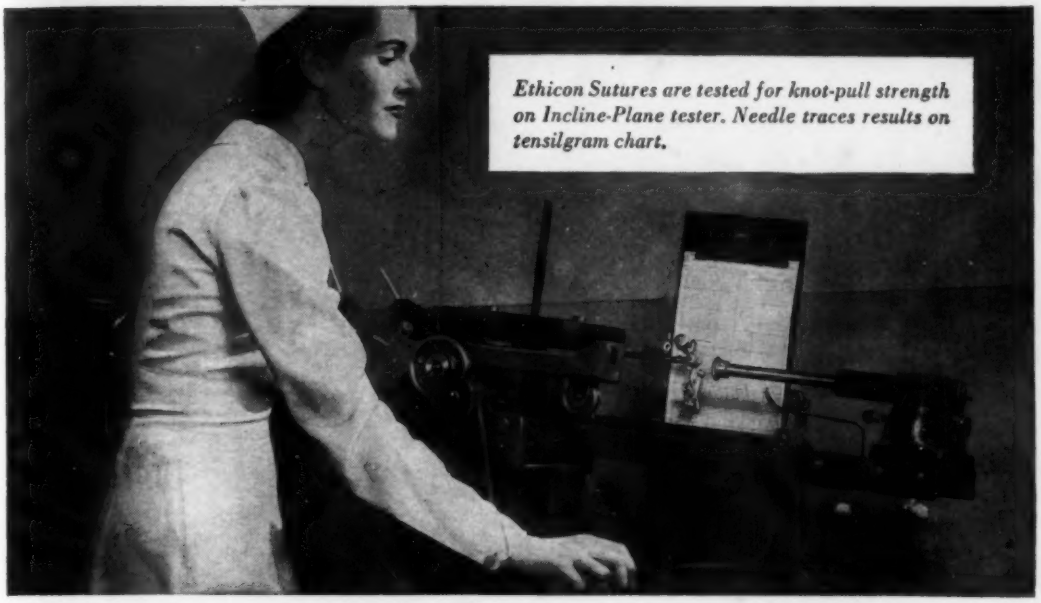
9. Hahn, P. F., and others: Peritoneal Absorption: Red Cells Labeled by Radio Iron Hemoglobin Move Promptly from Peritoneal Cavity into the Circulation, J. Exper. Med. **80**:77, 1944.

10. Wright, A. M., and Livingston, E. M.: The Leucocytosis of Internal Hemorrhage, New York State J. Med. **23**:286, 1923.

SUMMARY

Blood injected intraperitoneally is more rapidly absorbed if it is kept liquid by the use of an anticoagulant. Citrated blood is completely absorbed within twenty-four hours after injection. When uncitrated blood is injected, clots from 0.5 to 8 cm. in size are observed at four days but are absorbed within nine days after injection. Absorption of blood by way of the peritoneum will raise the blood volume to a maximum in twenty-four hours when 36 cc. per kilogram of body weight is injected in dogs. The leukocytosis following hemorrhage into the peritoneal cavity is in some way due to the presence of blood in the peritoneal cavity itself and is not accounted for on the basis of leukocytosis caused by hemorrhage. Moderate peritoneal thickening, omental fibrosis and mesothelial proliferation without adhesions follow repeated intraperitoneal injections of citrated autogenous blood.



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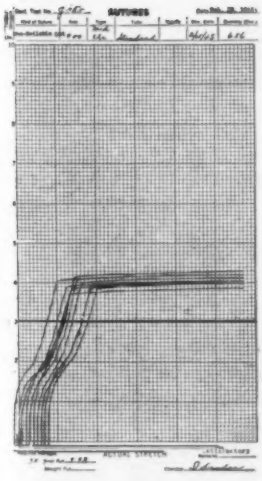

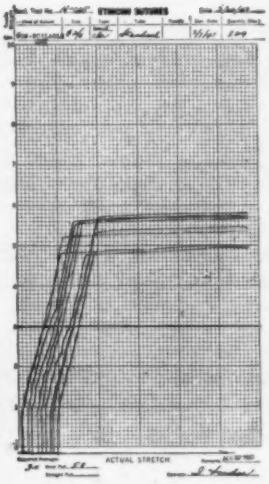

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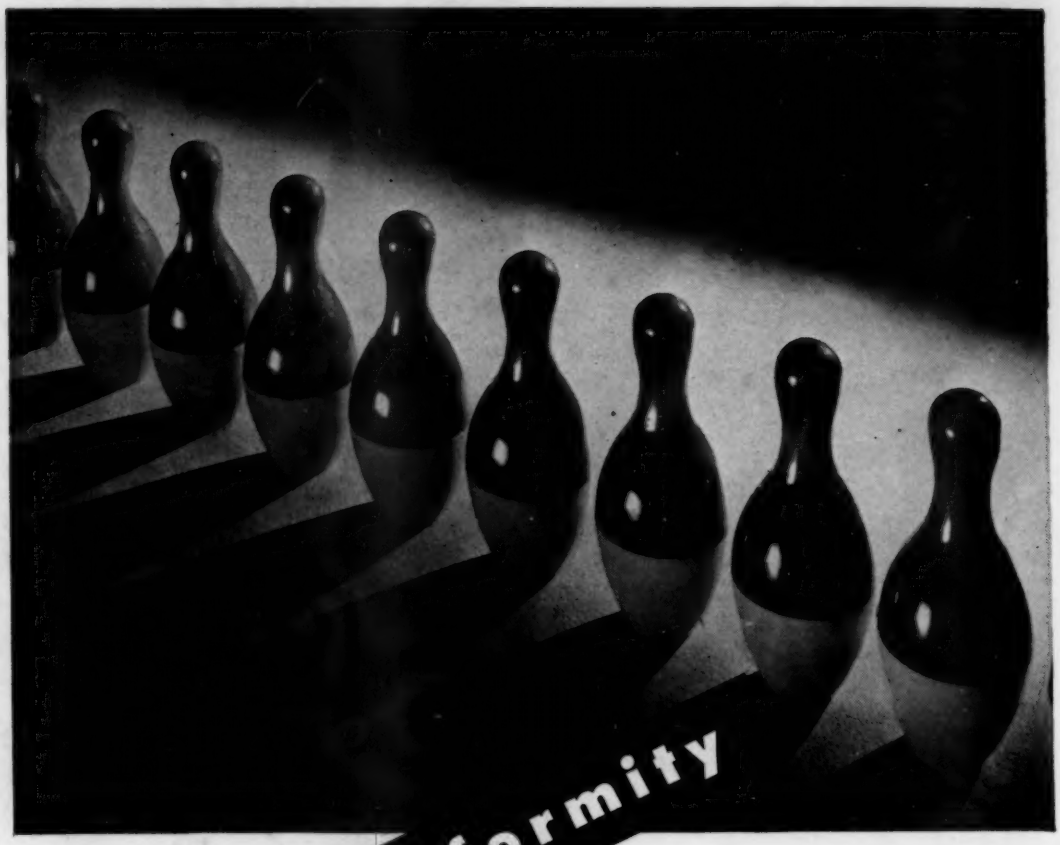

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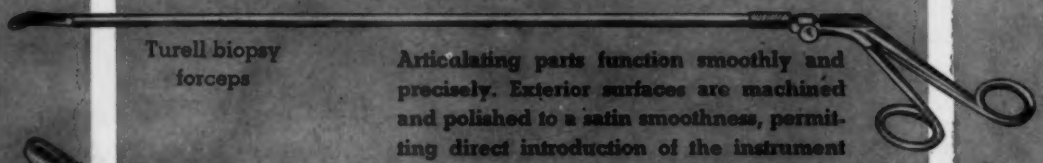

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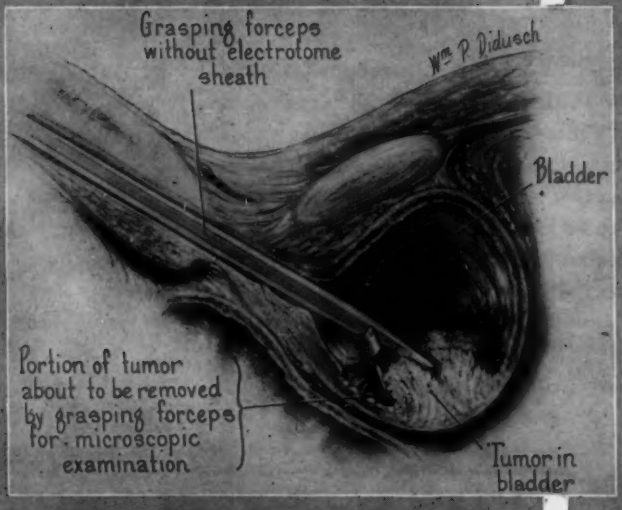
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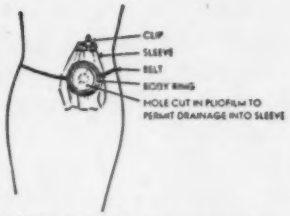

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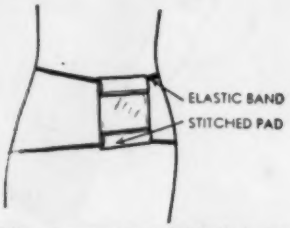

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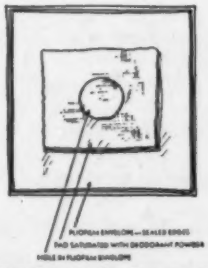

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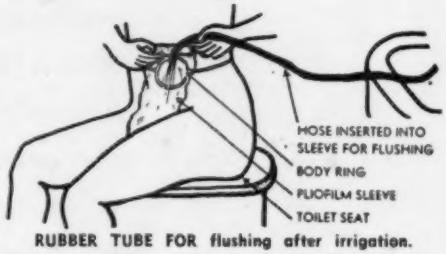

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